

# **Infectious Disease Epidemiology 2005 Annual Report**



**Office of Infectious Disease Services  
Bureau of Epidemiology and Disease Control  
Division of Public Health Services**

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Phoenix, AZ 85007**

**602-364-3676**



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## Executive Summary

The Infectious Disease Epidemiology Section (IDES) is one of five programs in the Office of Infectious Disease Services (OIDS) in the Bureau of Epidemiology and Disease Control. The Office of Infectious Disease Services in the Arizona Department of Health Services (ADHS) is responsible for monitoring and controlling diseases caused by infectious agents and toxins. The Office is also responsible for promulgating rules related to infectious disease surveillance, prevention, and control. The other four programs in the Office are: Tuberculosis Control, Hepatitis C Surveillance and Prevention, Sexually Transmitted Disease Control, and Vector-Borne and Zoonotic Diseases. HIV/AIDS surveillance and prevention activities are conducted by the Office of HIV/AIDS.

IDES is responsible for detecting, preventing, and controlling communicable diseases in several areas: foodborne, vaccine-preventable, nosocomial infections, and antibiotic resistant organisms. Program activities also cover other reportable infectious conditions that do not fit into these categories but are not covered by any of the other programs in the Office or Bureau.

The Program maintains a registry of over 70 notifiable communicable diseases; provides data and statistics on selected reportable infectious diseases by monitoring disease trends through surveillance and epidemiologic investigations; provides technical assistance to local and tribal health departments regarding prevention and control of disease; and provides information for health care providers and the public.

Some of the highlights for the period of January 1, 2005 through December 31, 2005 include:

- A statewide pertussis outbreak;
- A paralytic poliomyelitis case, the first identified in the U.S. since 1999 and the first ever identified imported vaccine-associated paralytic polio in the U.S.;
- Healthcare-associated streptococcal toxic shock syndrome;
- A measles case with international importation;
- Record levels of campylobacteriosis in Arizona;
- Development and implementation of investigation procedures for unexplained death surveillance; and
- Preparations for the transition to Arizona's web-based electronic surveillance system, the Medical Electronic Disease Surveillance Intelligence System (MEDSIS).
- The Program also conducted surveillance and provided infection control consultation for ADHS for the Phoenix shelter housing Hurricane Katrina evacuees in September 2005. The Hurricane Katrina Epidemiology Report is available separately at [http://www.azdhs.gov/phs/oids/data\\_reports.htm](http://www.azdhs.gov/phs/oids/data_reports.htm).

# I. Introduction



## **A. Data Sources and Limitations**

The Arizona Department of Health Services (ADHS) maintains registries of selected conditions that are reportable per Arizona Administrative Code R-9-202. The information is collected to assess and monitor the burden of disease, characterize affected populations, assess trends in disease occurrence, guide control efforts and evaluate prevention initiatives. The list of reportable conditions is based upon the list of Nationally Notifiable Infectious Diseases jointly developed by the Council of State and Territorial Epidemiologists (CSTE) and the Centers for Disease Control and Prevention (CDC). Additional conditions are included that are considered important for Arizona because of distinctions in the disease epidemiology or surveillance system in the state. The list is revised periodically to add newly emerging pathogens or remove conditions that are no longer considered relevant.

Public health surveillance case definitions are used to increase the specificity of reporting, and to allow comparability of diseases nationwide. Only cases meeting these standardized surveillance case definitions are included in the report. Criteria for surveillance case definitions are usually more stringent than those used by providers to diagnose and treat diseases.

State and local public health officials rely on health care providers, laboratories, hospitals and other facilities to report notifiable diseases or conditions. Local health jurisdictions submit case information to ADHS, which in turn reports case information without personal identifiers to CDC for purposes of compiling national statistics. Incomplete reporting is inherent to any passive surveillance system. Knowledge and awareness of current reporting rules, willingness to comply, severity of the disease, available diagnostic tests, age of the patient, confidentiality issues surrounding the disease, changes in the case definitions over time, and access to or availability of health care services all may influence the likelihood of reporting.

The 2005 population estimates (<http://www.azdhs.gov/plan/menu/info/pd.htm>) were used for rate calculations. Disease rates are calculated per 100,000 population unless otherwise specified and are not age-adjusted. Rate calculations based on a small number of reported cases or for counties with populations less than 100,000 are not considered reliable since they can be dramatically influenced by small changes in the number of reported cases.

## **B. Purpose of the Report**

The purpose of this report is to provide disease surveillance information to health care providers, health care organizations, governmental agencies, and other local health partners. This information is intended to assist agencies by providing uniform data on the disease burden in the state, trends in disease incidence and distribution and the evaluation of disease interventions.

Office staff collaborate with colleagues in the local and tribal health departments, as well as other ADHS Offices and Bureaus including: Environmental Health, Immunization Program Office, HIV/AIDS, State Health Laboratory Services, and Emergency Preparedness and Response within the Division of Public Health Services. Direct public health services, as they relate to surveillance, investigation, and response to infectious diseases of public health importance, are the responsibility of the 15 county health departments and tribal health departments and/or Indian Health Service Units. This report is designed to be utilized by external stakeholders in identifying trends, targeting prevention efforts, and determining resource needs. The Program would like to acknowledge both external and internal partners for their contributions to this report.

### **C. Reporting**

Arizona Administrative Code (AAC) R9-6-202, 203, 204, and 205 describe the morbidities required to be reported by health care providers, administrators of health care facilities, clinical laboratory directors, institutions, schools, pharmacists, and others.

On October 2, 2004, revisions to these sections of the AAC became effective. The 2004 Annual Report describes some of the rule changes. Though the changes occurred in 2004, the impact on the statistics for some conditions is probably better seen in the 2005 data, the first full year following the revisions. Tables outlining the reporting requirements are below. Additional information on the reporting requirements can be found on the Arizona Secretary of State's website at [http://www.azsos.gov/public\\_services/Title\\_09/9-06.pdf](http://www.azsos.gov/public_services/Title_09/9-06.pdf).



## D. Tables of Reportable Diseases

**Table 1. Reporting Requirements for a Health Care Provider or an Administrator of a Health Care Institution or Correctional Facility**

<input type="checkbox"/> *,O Amebiasis	<input type="checkbox"/> Hantavirus infection	<input type="checkbox"/> *,O Salmonellosis
<input checked="" type="checkbox"/> Anthrax	<input checked="" type="checkbox"/> Hemolytic uremic syndrome	<input type="checkbox"/> Scabies
<input type="checkbox"/> Aseptic meningitis: viral	<input type="checkbox"/> *,O Hepatitis A	<input checked="" type="checkbox"/> Severe acute respiratory syndrome
<input type="checkbox"/> Basidiobolomycosis	<input type="checkbox"/> Hepatitis B and D	<input type="checkbox"/> *,O Shigellosis
<input checked="" type="checkbox"/> Botulism	<input type="checkbox"/> Hepatitis C	<input checked="" type="checkbox"/> Smallpox
<input checked="" type="checkbox"/> Brucellosis	<input type="checkbox"/> *,O Hepatitis E	<input type="checkbox"/> Streptococcal Group A: Invasive disease
<input type="checkbox"/> *,O Campylobacteriosis	<input type="checkbox"/> Herpes genitalis	<input type="checkbox"/> Streptococcal Group B: Invasive disease in infants younger than 90 days of age
<input type="checkbox"/> Chancroid	<input type="checkbox"/> HIV infection and related disease	<input type="checkbox"/> <i>Streptococcus pneumoniae</i> (pneumococcal invasive disease)
<input type="checkbox"/> <i>Chlamydia</i> infection, genital	<input type="checkbox"/> Kawasaki syndrome	<input type="checkbox"/> Syphilis
<input checked="" type="checkbox"/> * Cholera	<input type="checkbox"/> Legionellosis (Legionnaires' disease)	<input type="checkbox"/> *,O Taeniasis
<input type="checkbox"/> Coccidioidomycosis (valley fever)	<input type="checkbox"/> Leptospirosis	<input type="checkbox"/> Tetanus
<input type="checkbox"/> Colorado tick fever	<input checked="" type="checkbox"/> Listeriosis	<input type="checkbox"/> Toxic shock syndrome
<input type="checkbox"/> Conjunctivitis: acute	<input type="checkbox"/> Lyme disease	<input type="checkbox"/> Trichinosis
<input type="checkbox"/> Creutzfeldt-Jakob disease	<input type="checkbox"/> Lymphocytic choriomeningitis	<input checked="" type="checkbox"/> Tuberculosis
<input type="checkbox"/> *,O Cryptosporidiosis	<input type="checkbox"/> Malaria	<input checked="" type="checkbox"/> Tuberculosis infection in a child younger than 6 (positive test result)
<input type="checkbox"/> <i>Cyclospora</i> infection	<input checked="" type="checkbox"/> Measles (rubeola)	<input checked="" type="checkbox"/> Tularemia
<input type="checkbox"/> Cysticercosis	<input checked="" type="checkbox"/> Meningococcal invasive disease	<input checked="" type="checkbox"/> Typhoid fever
<input type="checkbox"/> Dengue	<input checked="" type="checkbox"/> Mumps	<input checked="" type="checkbox"/> Typhus fever
<input type="checkbox"/> Diarrhea, nausea, or vomiting	<input checked="" type="checkbox"/> Pertussis (whooping cough)	<input checked="" type="checkbox"/> Unexplained death with a history of fever
<input checked="" type="checkbox"/> Diphtheria	<input checked="" type="checkbox"/> Plague	<input checked="" type="checkbox"/> Vaccinia-related adverse event
<input type="checkbox"/> Ehrlichiosis	<input checked="" type="checkbox"/> Poliomyelitis	<input type="checkbox"/> Vancomycin-resistant <i>Enterococcus</i> spp.
<input checked="" type="checkbox"/> Emerging or exotic disease	<input type="checkbox"/> Psittacosis (ornithosis)	<input checked="" type="checkbox"/> Vancomycin-resistant or Vancomycin-intermediate <i>Staphylococcus aureus</i>
<input checked="" type="checkbox"/> Encephalitis, viral or parasitic	<input checked="" type="checkbox"/> Q fever	<input checked="" type="checkbox"/> Vancomycin-resistant <i>Staphylococcus epidermidis</i>
<input checked="" type="checkbox"/> Enterohemorrhagic <i>Escherichia coli</i>	<input checked="" type="checkbox"/> Rabies in a human	<input type="checkbox"/> Varicella (chickenpox)
<input checked="" type="checkbox"/> Enterotoxigenic <i>Escherichia coli</i>	<input type="checkbox"/> Relapsing fever (borreliosis)	<input type="checkbox"/> *,O <i>Vibrio</i> infection
<input type="checkbox"/> *,O Giardiasis	<input type="checkbox"/> Reye syndrome	<input checked="" type="checkbox"/> Viral hemorrhagic fever
<input type="checkbox"/> Gonorrhea	<input type="checkbox"/> Rocky Mountain spotted fever	<input checked="" type="checkbox"/> West Nile virus infection
<input type="checkbox"/> <i>Haemophilus influenzae</i> : invasive disease	<input checked="" type="checkbox"/> * Rubella (German measles)	<input checked="" type="checkbox"/> Yellow fever
<input type="checkbox"/> Hansen's disease (Leprosy)	<input checked="" type="checkbox"/> Rubella syndrome, congenital	<input type="checkbox"/> *,O Yersiniosis

**Key:**

- Submit a report by telephone or through an electronic reporting system authorized by the Department within 24 hours after a case or suspect case is diagnosed, treated, or detected or an occurrence is detected.
- \* If a case or suspect case is a food handler or works in a child care establishment or a health care institution, instead of reporting within the general reporting deadline, submit a report within 24 hours after the case or suspect case is diagnosed, treated, or detected.
- Submit a report within one working day after a case or suspect case is diagnosed, treated, or detected.
- Submit a report within five working days after a case or suspect case is diagnosed, treated, or detected.
- Submit a report within 24 hours after detecting an outbreak.

**Table 2. Reporting Requirements for an Administrator of a School, Child Care Establishment, or Shelter**

<input type="checkbox"/>	Campylobacteriosis	<input type="checkbox"/>	Mumps
<input type="checkbox"/>	Conjunctivitis: acute	<input type="checkbox"/>	Pertussis (whooping cough)
<input type="checkbox"/>	Cryptosporidiosis	<input type="checkbox"/>	Rubella (German measles)
<input type="checkbox"/>	Diarrhea, nausea, or vomiting	<input type="checkbox"/>	Salmonellosis
<input type="checkbox"/>	Enterohemorrhagic <i>Escherichia coli</i>	<input type="checkbox"/>	Scabies
<input type="checkbox"/>	<i>Haemophilus influenzae</i> : invasive disease	<input type="checkbox"/>	Shigellosis
<input type="checkbox"/>	Hepatitis A	<input type="checkbox"/>	Streptococcal Group A infection
<input type="checkbox"/>	Measles	<input type="checkbox"/>	Varicella (chicken pox)
<input type="checkbox"/>	Meningococcal invasive disease		

**Key:**

- Submit a report within 24 hours after detecting a case or suspect case.
- Submit a report within five working days after detecting a case or suspect case.
- Submit a report within 24 hours after detecting an outbreak.

**Table 3. Clinical Laboratory Director Reporting Requirements**

①	Arboviruses	Ⓢ, ⊕	<i>Haemophilus influenzae</i> , type B, isolated from a normally sterile site	Ⓢ+	Respiratory syncytial virus
Ⓢ, Ⓢ, ⊕	<i>Bacillus anthracis</i>	Ⓢ, ⊕	<i>Haemophilus influenzae</i> , other, isolated from a normally sterile site	①, ⊕	<i>Salmonella</i> spp.
Ⓢ, ⊕	<i>Bordetella pertussis</i>	Ⓢ	Hantavirus	Ⓢ	SARS-associated corona virus
①, ⊕	<i>Brucella</i> spp.	Ⓢ	Hepatitis A virus (anti-HAV-IgM serologies)	①, ⊕	<i>Shigella</i> spp.
Ⓢ	<i>Campylobacter</i> spp.	Ⓢ	Hepatitis B virus (anti-Hepatitis B core-IgM serologies, Hepatitis B surface antigen serologies, and detection of viral nucleic acid)	Ⓢ, ⊕	<i>Streptococcus</i> Group A, isolated from a normally sterile site
Ⓢ	CD <sub>4</sub> -T-lymphocyte count of fewer than 200 per microliter of whole blood or CD <sub>4</sub> -T-lymphocyte percentage of total lymphocytes of less than 14%	Ⓢ	Hepatitis C virus	Ⓢ	<i>Streptococcus</i> Group B, isolated from a normally sterile site in an infant younger than 90 days of age
Ⓢ	<i>Chlamydia trachomatis</i>	Ⓢ	Hepatitis D virus	Ⓢ, ⊕	<i>Streptococcus pneumoniae</i> and its drug sensitivity pattern, isolated from a normally sterile site
Ⓢ, Ⓢ	<i>Clostridium botulinum</i> toxin (botulism)	Ⓢ	Hepatitis E virus	Ⓢ	<i>Treponema pallidum</i> (syphilis)
Ⓢ	<i>Coccidioides</i> spp., by culture or serologies	Ⓢ	HIV (by culture, antigen, antibodies to the virus, or detection of viral nucleic acid)	Ⓢ	Vancomycin-resistant <i>Enterococcus</i> spp.
①	<i>Coxiella burnetii</i>	Ⓢ	HIV—any test result for an infant (by culture, antigen, antibodies to the virus, or detection of viral nucleic acid)	①, ⊕	Vancomycin-resistant or Vancomycin-intermediate <i>Staphylococcus aureus</i>
Ⓢ	<i>Cryptosporidium</i> spp.	Ⓢ+	Influenza virus	①, ⊕	Vancomycin-resistant <i>Staphylococcus epidermidis</i>
①	<i>Cyclospora</i> spp.	Ⓢ, ⊕	<i>Legionella</i> spp. (culture or DFA)	Ⓢ, Ⓢ	Variola virus (smallpox)
Ⓢ, Ⓢ	Dengue virus	①, ⊕	<i>Listeria</i> spp., isolated from a normally sterile site	①, ⊕	<i>Vibrio</i> spp.
Ⓢ, Ⓢ	Emerging or exotic disease agent	Ⓢ <sup>1</sup>	Methicillin-resistant <i>Staphylococcus aureus</i> , isolated from a normally sterile site	Ⓢ, Ⓢ	Viral hemorrhagic fever agent
Ⓢ	<i>Entamoeba histolytica</i>	Ⓢ, ⊕ <sup>2</sup>	<i>Mycobacterium tuberculosis</i> complex and its drug sensitivity pattern	①	West Nile virus
①	<i>Escherichia coli</i> O157:H7	Ⓢ	<i>Neisseria gonorrhoeae</i>	①, ⊕	<i>Yersinia</i> spp. (other than <i>Y. pestis</i> )
①, ⊕	<i>Escherichia coli</i> , Shiga-toxin producing	Ⓢ, ⊕	<i>Neisseria meningitidis</i> , isolated from a normally sterile site	Ⓢ, Ⓢ, ⊕	<i>Yersinia pestis</i> (plague)
Ⓢ, Ⓢ, ⊕	<i>Francisella tularensis</i>	Ⓢ	<i>Plasmodium</i> spp.		

**Key:**

Ⓢ Submit a report immediately after receiving one specimen for detection of the agent. Report receipt of subsequent specimens within five working days after receipt.

Ⓢ Submit a report within 24 hours after obtaining a positive test result.

① Submit a report within one working day after obtaining a positive test result.

Ⓢ Submit a report within five working days after obtaining a positive test result or a test result specified in Table 3.

Ⓢ Submit an isolate of the organism for each positive culture to the Arizona State Laboratory at least once each week, as applicable.

+ A clinical laboratory director may report aggregate numbers of positive test results every five working days rather than submitting individual reports as required in R9-6-204(B).

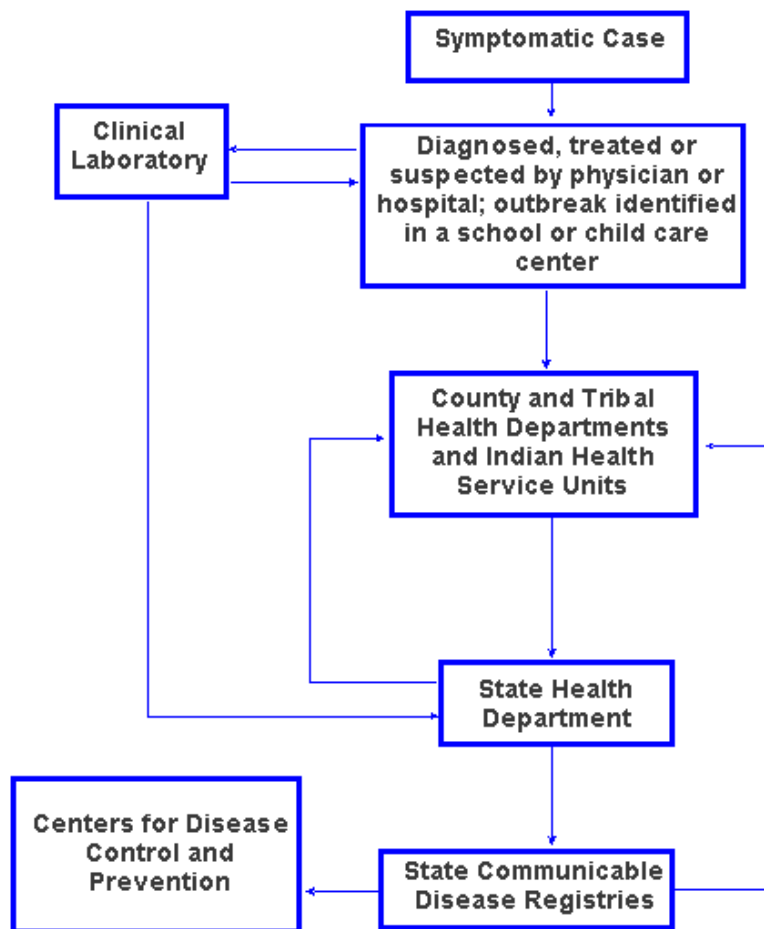
<sup>1</sup> Submit a report only when an initial positive result is obtained for an individual.

<sup>2</sup> Submit an isolate of the organism only when an initial positive result is obtained for an individual, when a change in resistance pattern is detected, or when a positive result is obtained ≥ 12 months after the initial positive result is obtained for an individual.

Arizona requires reporting by both health care providers and clinical laboratories as a dual surveillance measure to increase the sensitivity of the surveillance system and improve the completeness of reporting. Diseases are reported via fax, mail, telephone, or electronic systems using the communicable disease report (CDR) form. Additional information on communicable disease reporting as well as reporting and investigation forms can be found on the Department's website at: [http://www.azdhs.gov/phs/oids/dis\\_rpt.htm](http://www.azdhs.gov/phs/oids/dis_rpt.htm).

Since local health departments are the primary response agency, health care providers report notifiable conditions to the local health departments for immediate investigation and initiation of control measures, as needed. Figure 1 outlines the reporting structure and flow of information in Arizona.

**Figure 1. Flow of communicable disease reports**



All information supplied to state or county public health agencies is maintained in strict confidentiality in conformance to state statutes.

# E.State and County Health Department Contact Information

## Arizona Department of Health Services

### Infectious Disease Epidemiology Section

150 N. 18<sup>th</sup> Avenue Suite 140  
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Phone: (602) 364-3676  
Fax: (602) 364-3119

### Emergency Answering Service

Phone: (480) 303-1919

### State Laboratory Services

250 N. 17<sup>th</sup> Avenue  
Phoenix, AZ 85007-3231  
Phone: (602) 542-1188  
Fax: (602) 542-1169

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## County Health Departments

### Apache County Health Department

395 South 1<sup>st</sup> Street West  
PO Box 697  
St. Johns, AZ 85936  
Phone: (928) 337-4364  
Fax: (928) 337-2062

### Greenlee County Health Department

PO Box 936  
5<sup>th</sup> & Leonard Streets  
Clifton, AZ 85533  
Phone: (928) 865-2601  
Fax: (928) 865-1929

### Cochise County Health Department

1415 W. Melody Lane, Bldg A.  
Bisbee, AZ 85603-3090  
Phone: (520) 432-9400  
Fax: (520) 432-9480

### La Paz County Health Department

1112 Joshua Street #206  
Parker, AZ 85344  
Phone: (928) 669-1100  
Fax: (928) 669-6703

### Coconino County Department of Health Services

2625 N. King Street  
Flagstaff, AZ 86004  
Phone: (928) 522-7800  
Fax: (928) 522-7808

### Maricopa County Department of Public Health

4041 N. Central Ave Suite 1400  
Phoenix, AZ 85012  
Phone: (602) 506-6900  
Fax: (602) 506-6885

### Gila County Health Department

1400 E. Ash  
Globe, AZ 85501  
Phone: (928) 425-3231  
Fax: (928) 425-0794

### Mohave County Health Department

PO Box 7000  
318 N. 5<sup>th</sup> Street  
Kingman, AZ 86401-7000  
Phone: (928) 753-0743  
Fax: (928) 718-5547

### Graham County Health Department

826 W. Main  
Safford, AZ 85546  
Phone: (928) 428-0110  
Fax: (928) 428-8074

### Navajo County Health Services District

117 E. Buffalo Street  
Holbrook, AZ 86025  
Phone: (928) 524-4750  
Fax: (928) 524-4759

**Pima County Health Department**

150 W. Congress Street #334  
Tucson, AZ 85701  
Phone: (520) 740-8261  
Fax: (520) 791-0366

**Pinal County Health Department**

500 South Main  
PO Box 2945  
Florence, AZ 85232-2945  
Phone: (520) 866-7319  
Fax: (520) 866-7310

**Santa Cruz County Health Department**

2150 N. Congress  
Nogales, AZ 85621  
Phone: (520) 375-7900  
Fax: (520) 761-4813

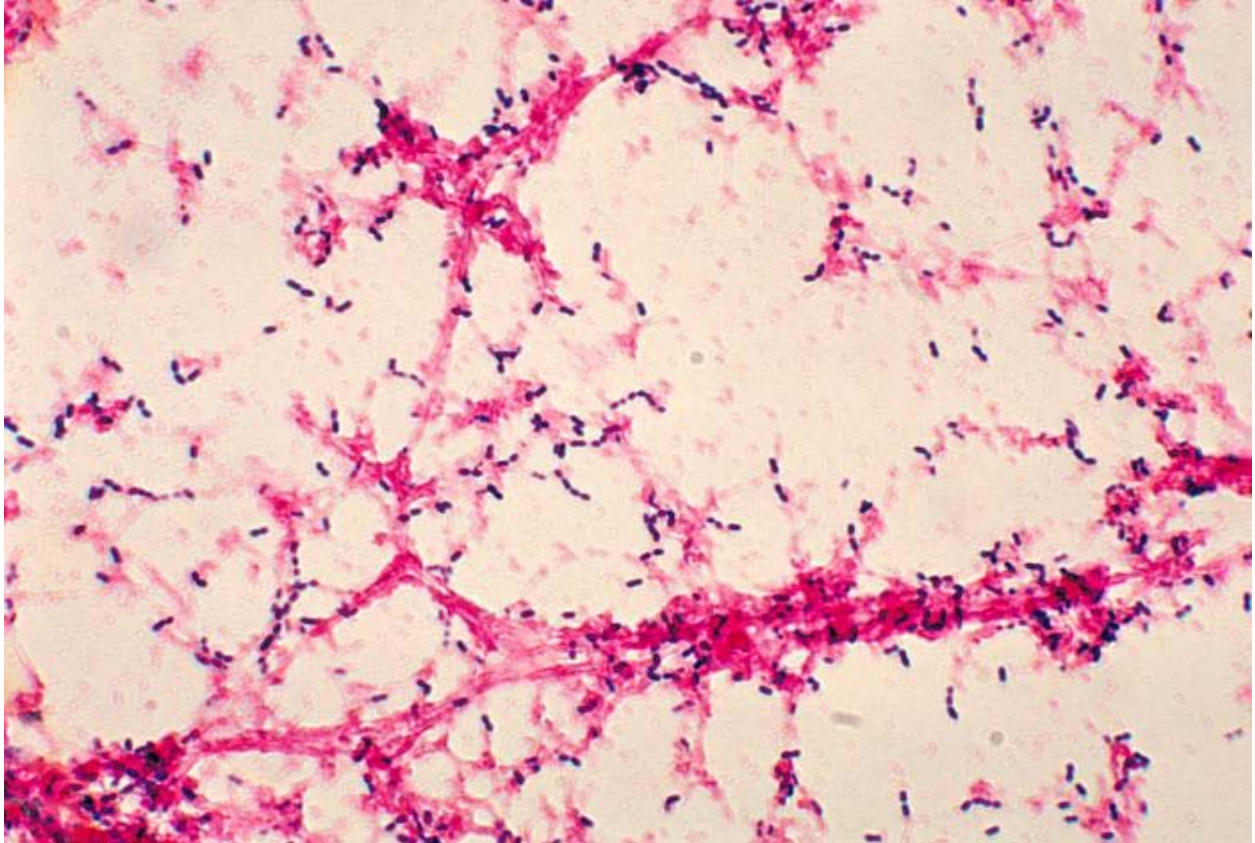
**Yavapai County Health Department**

1090 Commerce Drive  
Prescott, AZ 86305  
Phone: (928) 771-3122  
Fax: (928) 771-3369

**Yuma County Health Department**

2200 W. 28<sup>th</sup> Street Suite #137  
Yuma, AZ 85364  
Phone: (928) 317-4550  
Fax: (928) 317-4591

## II. Disease Statistics



*Streptococcus pneumoniae*

## **A. Population Estimates for 2005**

Office of Vital Statistics, Arizona Department of Health Services  
<http://www.azdhs.gov/plan/menu/info/pop/pop05/pd05.htm>

## **B. Tables of Cases and Rates of Reportable Diseases**

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8. [Rates of Reported Cases of Selected Notifiable Diseases by Race/Ethnicity, 2005](#)
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10. [Rates of Reported Cases of Selected Notifiable Diseases by County, 5 Year Age Groupings, and Gender, 2005](#)



### III. Disease Summaries

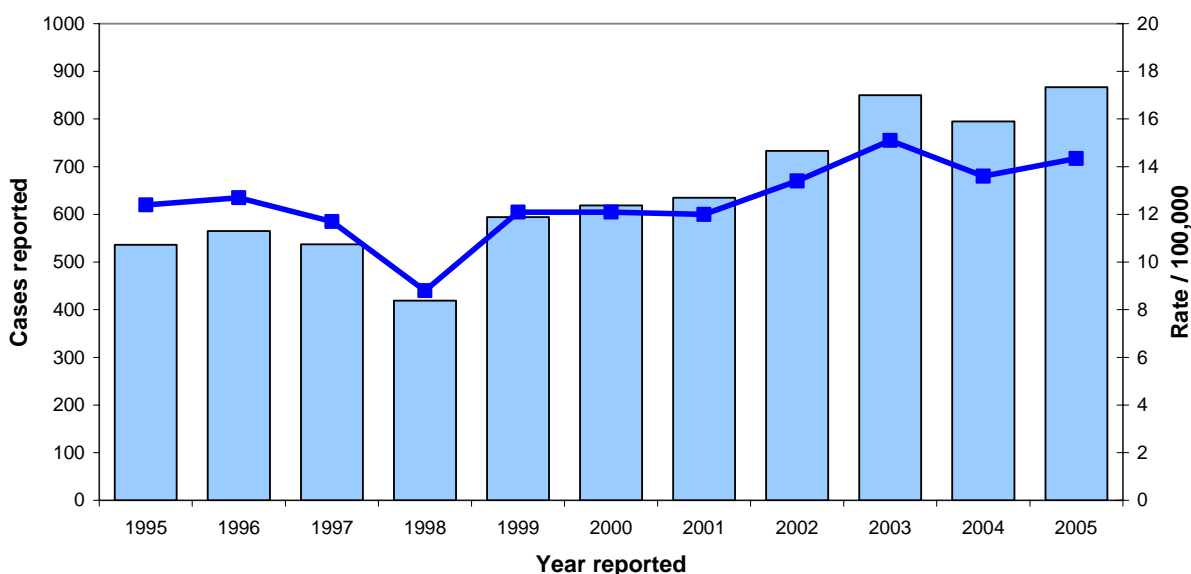


## A. Campylobacteriosis

Gastrointestinal illnesses caused by the bacteria *Campylobacter* spp. are among the classic foodborne illnesses, with infection mainly attributed to consumption of contaminated food or water. In recent years, the number of reported human cases of foodborne illness caused by *Campylobacter* has increased in the United States and Arizona, demonstrating the need for careful review of the sources of these common bacteria.

Over the past ten years, the number and rate of reported cases of campylobacteriosis has increased in Arizona (Figure 2), with 536 cases reported in 1995 (12.0 per 100,000 population) and 867 in 2005 (14.3 per 100,000 population). During this time, *Campylobacter* has been gaining recognition as an important cause of foodborne illness. Until the development of a selective medium in 1977, these organisms were known mainly by veterinarians as animal pathogens.<sup>1</sup> Since the development of more sophisticated isolation techniques in 1972 and 1989, the true disease burden of these organisms has become more apparent.<sup>2</sup> Reported cases and rates in Figure 2 represent surveillance of campylobacteriosis for ten consecutive years.

**Figure 2. Reported campylobacteriosis, Arizona, 1995-2005**

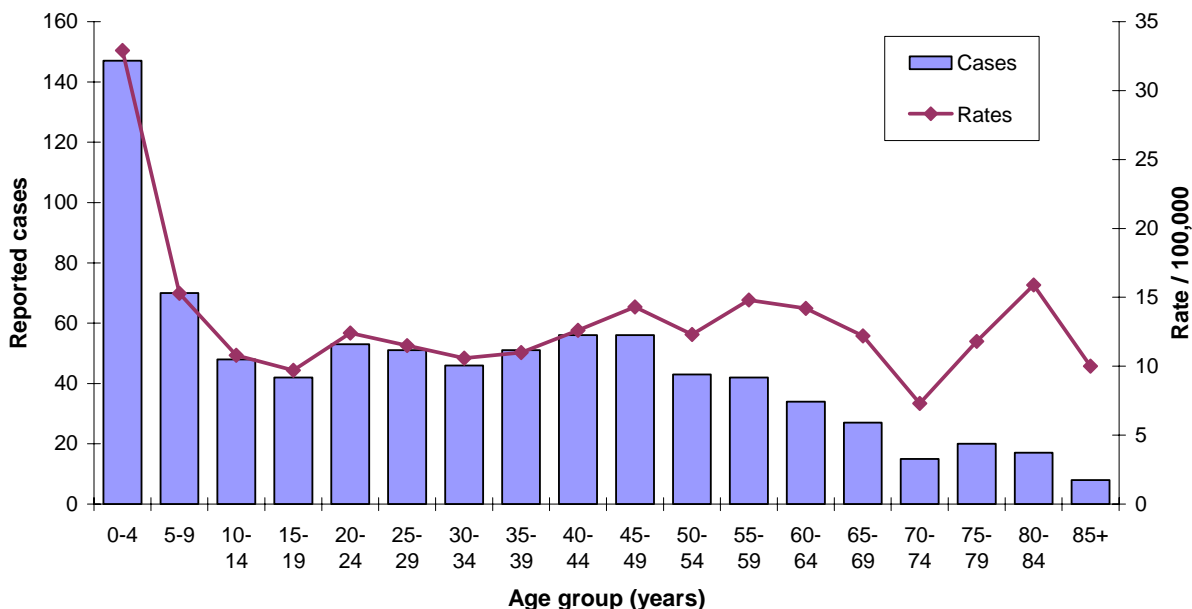


Reported cases of campylobacteriosis are highest among young children, both in Arizona and nationally. In 2005, the rate among 0-4 year olds was 32.9 per 100,000 which is twice the rate observed in any of the other age group (Figure 3). Campylobacteriosis cases also vary widely by geographic region. The incidence of reported cases in Arizona in 2005 was highest in the northeast counties, with reported rates 2 to 6 times higher than the statewide rate of 14.3 per 100,000 (Figure 4).

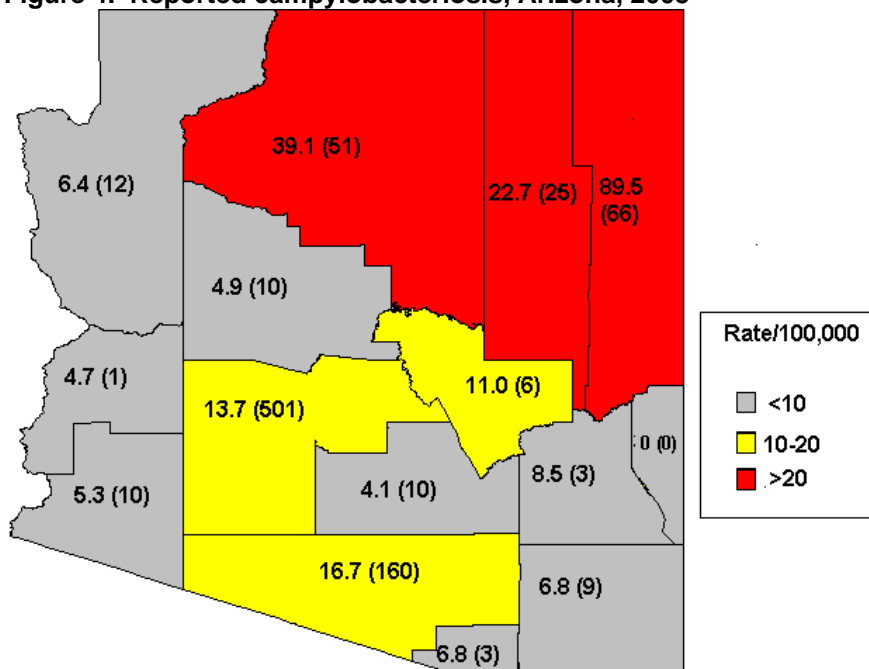
<sup>1</sup> Mosenthal AC, RL Mones, and VD Bokkenheuser. *Campylobacter fetus jejuni* enteritis; in New York City. N Y State Journal of Medicine. 1981. 81(3):321-323.

<sup>2</sup> Humphrey TJ. An appraisal of the efficacy of pre-enrichment for the isolation of *Campylobacter jejuni* from water and food. Journal of Applied Bacteriology 1989. 66(2):119-126.

**Figure 3. Age group of reported campylobacteriosis cases, Arizona, 2005**



**Figure 4. Reported campylobacteriosis, Arizona, 2005**



Unlike other enteric pathogens such as *Shigella* spp., person-to-person transmission via the fecal-oral route does not appear to be significant for *Campylobacter* spp. A small percentage of people infected with *Campylobacter* acquire the bacteria from direct contact with animals which commonly harbor the bacteria in their gastrointestinal tract, such as wild and domestic ruminants, swine, dogs, cats, fowl, and rodents. Only two species of *Campylobacter* are known to cause illness in humans, and the environments where they live can shed light on the main sources of infection. *Campylobacter jejuni* is responsible for the majority of human cases and has been shown to be most prevalent in poultry, while pigs appear to be the preferred niche of

*C. coli*, the second most common species. Researchers in Turkey have found that as much as 85% of poultry sold in the grocery store is contaminated with *C. jejuni*.<sup>3</sup> One example of the impact contaminated poultry has on the rates of campylobacteriosis was shown in 1999 when poultry was removed from the market in Belgium due to concerns that it might be contaminated with dioxins. Rates of *Campylobacter* infection fell by 40 percent during this ban and then returned to the previous level after chicken was put back on the shelves.<sup>4</sup>

Several public education campaigns have been released recently in parts of the U.S. educating consumers about the importance of keeping raw poultry separate from ready-to-eat products to avoid cross-contamination. Although expanding public education may be effective in altering the incidence of *Campylobacter* infections in kitchens, many researchers are calling on the poultry industry and the USDA to determine new, more efficient ways to rid poultry of bacteria such as *Campylobacter* before it reaches the shelves of grocery stores. Decidedly, it is currently a two-pronged effort: regulators need to ensure food is safe for consumption while consumers take care to prepare and store food properly.

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<sup>3</sup> Sava M and Ozdemir H. Prevalence of thermophilic *Campylobacter* spp. in retail chicken meat in Ankara. *Journal of Food Safety*. 2006. 26(3):244.

<sup>4</sup> Vellinga A and van Loock F. The dioxin crisis as experiment to determine poultry-related *Campylobacter* enteritis. *Emerging Infectious Diseases*. 2002 8(2):19-22.

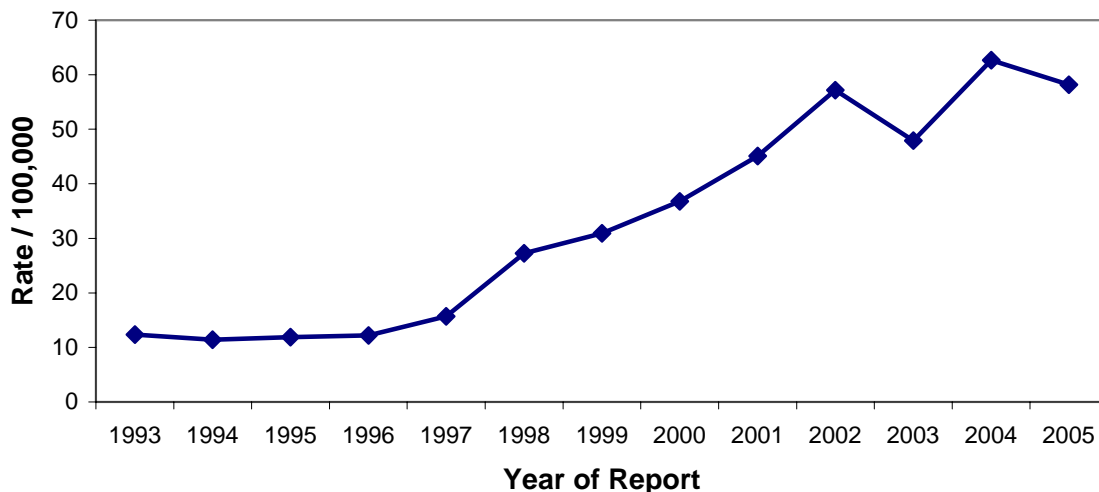
## B. Coccidioidomycosis

Since 1995, coccidioidomycosis has been a nationally reportable disease at the southwest regional level and includes a requirement for laboratory confirmation. Arizona began requiring mandatory laboratory reporting in 1997, which led to an increase in the number of reported cases. Subsequently, added benefits of improved timeliness and completeness of reporting have been noted.

The number of cases reported continued to steadily increase after 1997, and this increase is not likely to be simply associated with improved methods of reporting.<sup>5</sup> Several potential explanations may be offered to account for the increase, including the large number of susceptible individuals moving into a naturally endemic area with no prior history of exposure. Another factor may be increased awareness among both the general public and physicians, leading to more requests for laboratory testing for *Coccidioides* species. Urban sprawl and construction may add to the generation of dust-containing spores. And, a recent study also implies that a wet summer in the preceding one to two years followed by a dry winter may lead to an increase in coccidioidomycosis.<sup>6</sup> One of these factors, or more likely a combination of several, may help explain why the number and rate of cases over the past ten years have continued to rise.

To date, the highest number of cases ever reported in Arizona was in 2004. In 2004, a total of 3,665 cases of coccidioidomycosis were reported in Arizona, with a rate of 62.8 cases per 100,000 Arizona residents, a 281% increase since 1997. In 2005, the number reported was similarly high with 3,515 cases of coccidioidomycosis, with a rate of 58.2 cases per 100,000 Arizona residents. Although the incidence of newly reported cases is slightly lower in 2005 than in 2004, 2005 is the second highest year for coccidioidomycosis since reporting began (Figure 5).

Figure 5. Rates of reported coccidioidomycosis in Arizona, 1993-2005

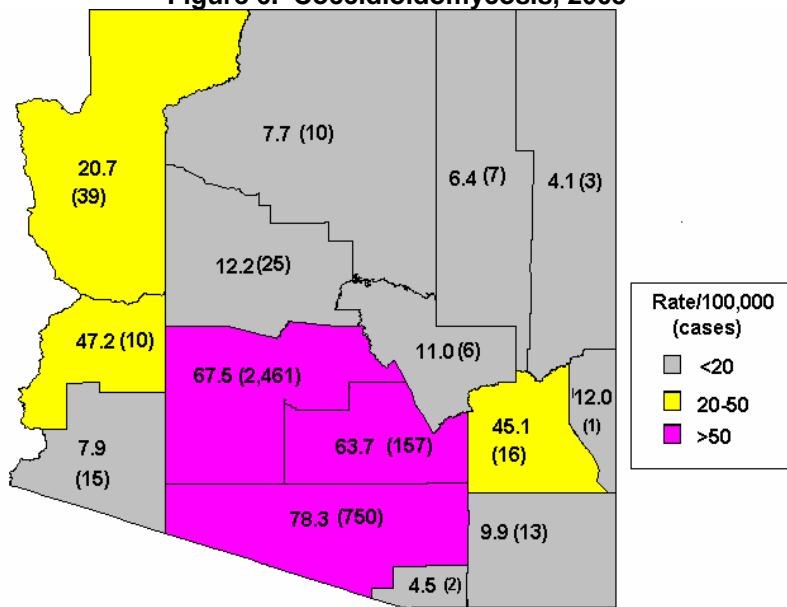


<sup>5</sup> CDC. Increase in Coccidioidomycosis – Arizona, 1998-2001, 2003. MMWR 2003; 52:109-112.

<sup>6</sup> Comrie, Andrew. Climate factors influencing coccidioidomycosis seasonality and outbreaks. Environmental Health Perspectives. June 2005; 113(6): 688-692.

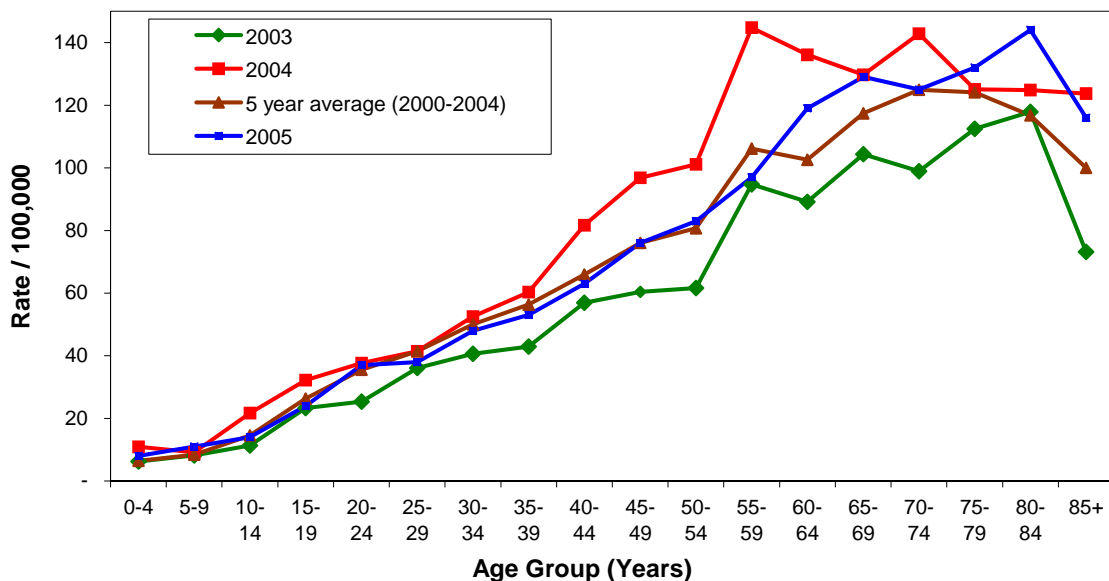
In 2005, the incidence rate in males (63.9 per 100,000) continued to be higher than in females (52.0 per 100,000), which may be due to factors such as occupational exposure and duration of outdoor activities. Incidence rate by county reveals the highest rate in Pima (78.3 per 100,000), followed by Maricopa (67.5 per 100,000), and Pinal (63.7 per 100,000) (Figure 6). These three counties consistently have not only the highest number of cases, but also the highest rates. Cases reported in 2005 by county are: Maricopa (2,461), Pima (750), Pinal (157).

**Figure 6. Coccidioidomycosis, 2005**



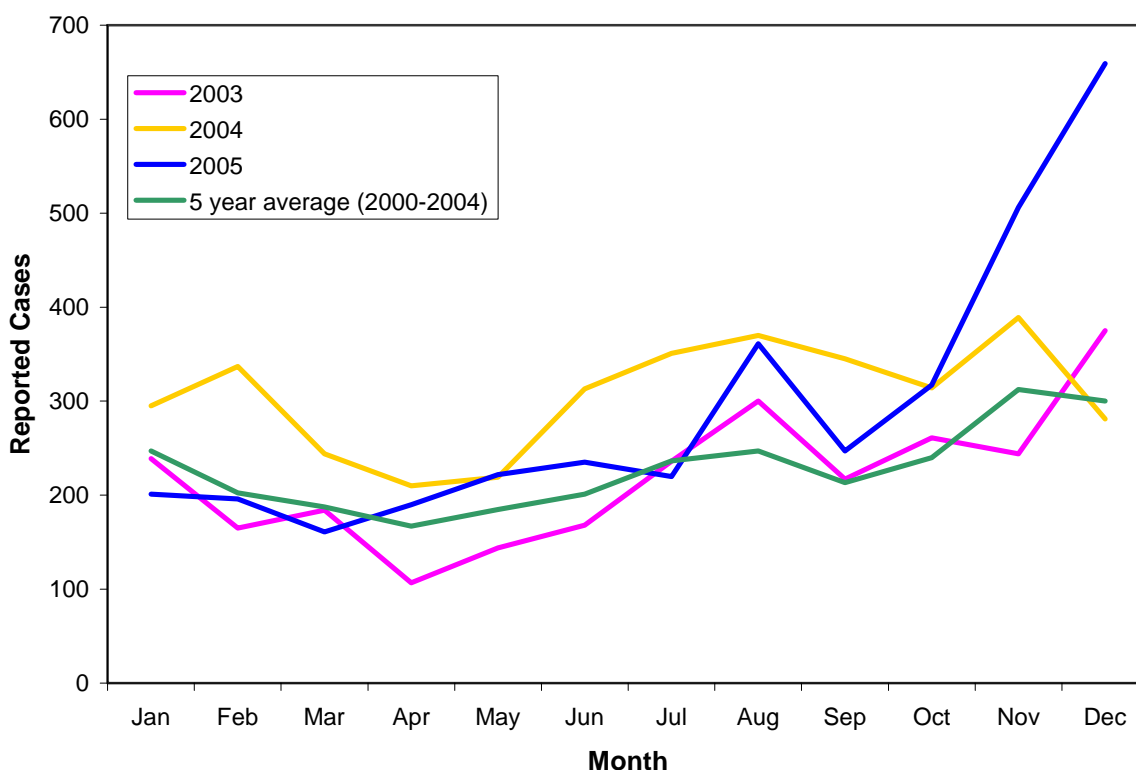
Age-specific rates for 2005 show similar trends to past years, with incidence rates highest in persons aged  $\geq 55$  years (Figure 7). Age is a risk factor for developing symptoms and persons in older age groups are therefore most likely to be diagnosed.

**Figure 7. Coccidioidomycosis rates per 100,000, by age and year, Arizona, 2000-2005**



Disease incidence in Arizona appears to peak in the winter during the months of November to February, with a smaller rise noted in late summer between July and September (Figure 8). This trend varies from southern California, where, in an earlier study, infection rates from coccidioidomycosis were higher in late summer and early fall.<sup>7</sup> During 2005, reported Arizona cases of coccidioidomycosis displayed an increase in August; however, the number of new cases reported in August still remained slightly below that recorded in 2004. Most noticeably, a rapid and substantial rise in new coccidioidomycosis cases was reported beginning in October and continuing through the end of 2005. Several factors may have contributed to this increase. Greater public awareness of the disease and a physician education notice to consider coccidioidomycosis when presented with certain respiratory syndromes may have led to increased testing and diagnosis, which in turn could lead to an increase in new cases being reported that might have otherwise been left undiagnosed. Also, a change in the state surveillance system resulted in many cases that would have been counted as January cases being reported as December cases. These factors alone, however, cannot explain entirely the large increase in cases that occurred in the latter half of 2005 and continues into 2006. Further studies and educational efforts are planned for 2006 to better understand and characterize this recent increase.

**Figure 8. Reported coccidioidomycosis cases by month, Arizona, 2000-2005**



Most infections of *Coccidioides* are sub-clinical or self-limited. Clinical manifestations range from influenza-like illness to severe pneumonia and, more rarely, extra-pulmonary or

<sup>7</sup> Smith CE, Beard RR, Whiting EG, Rosenberg HG. Effect of season and dust control on coccidioidomycosis. JAMA. 1946; 132:833-8.

disseminated disease. It is important to note, that hospitalizations associated with a diagnosis of coccidioidomycosis have substantially increased from 1998, indicating an increase in the number of cases that present with severe disease. However, the number of deaths attributed to coccidioidomycosis in recent years has been relatively consistent, even as the rate of disease incidence has increased. Health-care providers in Arizona may want to consider coccidioidomycosis in the differential diagnosis of patients with influenza-like illness given that the peak activity of influenza and coccidioidomycosis coincides.<sup>8</sup> Recommendations have also been issued to consider testing for *Coccidioides* species when a diagnosis of community-acquired pneumonia (CAP) is given.<sup>9</sup>

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<sup>8</sup> Smith CE, Beard RR, Whiting EG, Rosenberg HG. Effect of season and dust control on coccidioidomycosis. JAMA. 1946; 132:833-8.

<sup>9</sup> ADHS. Prevention Bulletin. March/April 2006; Vol. 20, No. 2: 6.

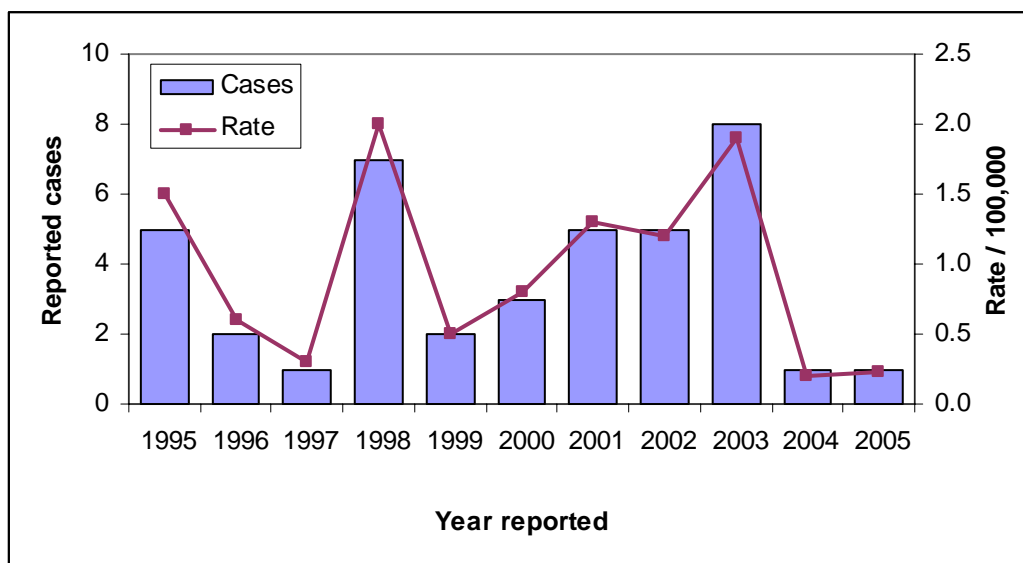


### C. Invasive *Haemophilus influenzae*

Invasive *Haemophilus influenzae* disease results in severe infections, particularly among infants and young children. There are six encapsulated types of *Haemophilus influenzae*: A, B, C, D, and F. An unencapsulated form also causes invasive disease, but is generally less virulent than encapsulated strains and rarely causes serious illness. The most severe infections in infants and young children are caused by *Haemophilus influenzae* serotype b (Hib). The most common types of invasive disease caused by Hib include meningitis, epiglottitis, septic arthritis, pneumonia, and cellulitis. Before the introduction of Hib vaccine in 1985, invasive Hib disease was the leading cause of bacterial meningitis and other invasive bacterial disease among children younger than 5 years of age, and approximately two thirds of cases occurred among children younger than 18 months of age.<sup>10</sup> The most striking feature of Hib disease is age-dependent susceptibility, as it is uncommon beyond five years of age.

In Arizona, very few cases of Hib in children less than five are reported each year, and these are mainly in unvaccinated or undervaccinated infants and children. During 2005, one case of invasive Hib disease was reported in a child less than five years of age with unknown vaccination history (Figure 9).

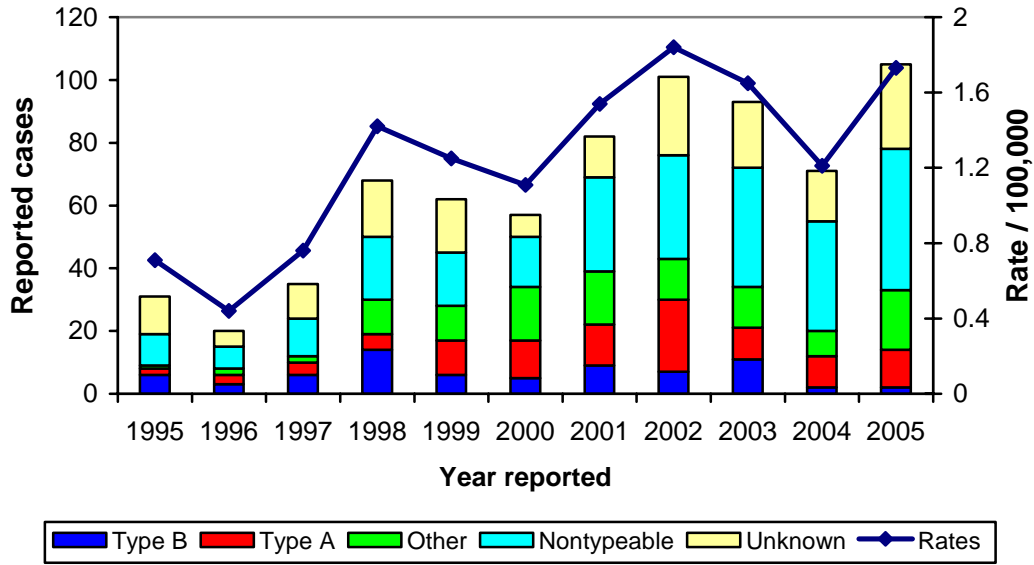
**Figure 9. Reported invasive *Haemophilus influenzae* type B in children aged <5 years, Arizona, 1995-2005**



In Arizona, all invasive disease caused by *Haemophilus influenzae* is reportable, and all *H. influenzae* isolates from sterile sites are required to be forwarded to the State Laboratory for serotyping. From 1995 through 2005, *H. flu* rates have been steadily increasing from 0.41 cases per 100,000 persons in 1995 to 1.73 cases per 100,000 persons in 2005. In addition, the proportion of invasive *H. influenzae* disease caused by nontypeable strains has increased (Figure 10).

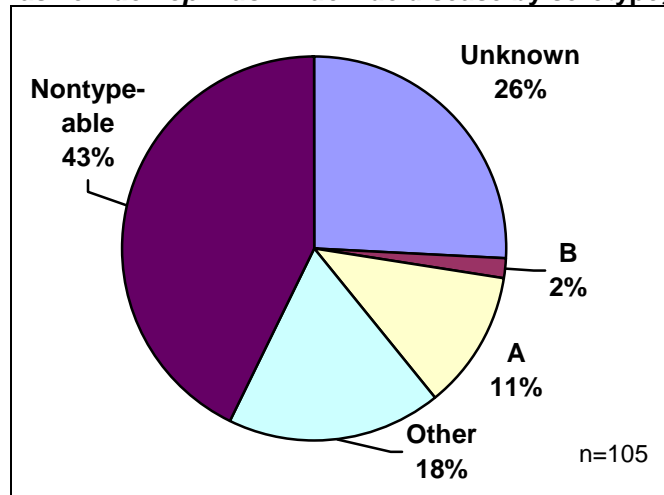
<sup>10</sup> Centers for Disease Control and Prevention. Epidemiology and Prevention of Vaccine-Preventable Diseases. Atkinson W, Hamborsky J, McIntyre L, Wolfe S, eds. 9th ed. Washington DC: Public Health Foundation, 2006.

Figure 10. Reported invasive *Haemophilus influenzae* disease, Arizona, 1995-2005



During 2005, 105 cases of invasive *Haemophilus influenzae* disease were reported. Of the 31 case isolates with serotypes other than B, 12 isolates were serotype A, 9 were serotype E, and eight were serotype D. Forty-five isolates were nontypeable (unencapsulated) strains (Figure 11).

Figure 11. Invasive *Haemophilus influenzae* disease by serotype, Arizona, 2005



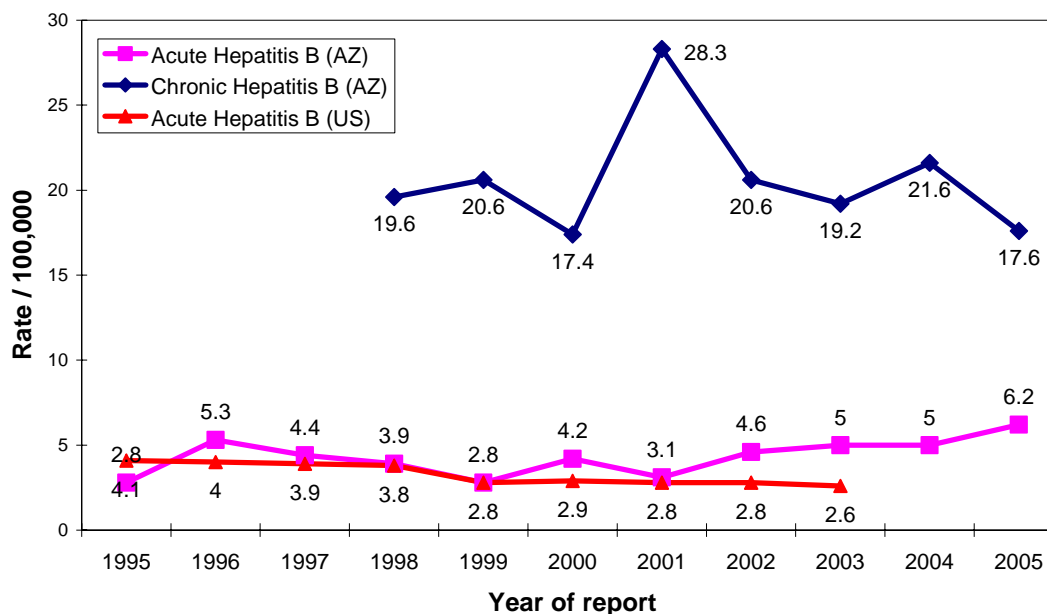
## D. Hepatitis B

In 2005, 375 cases of acute hepatitis B (6.2/100,000 population) and 1,064 newly reported cases of chronic hepatitis B (17.6/100,000 population) were reported in Arizona. Acute hepatitis B has been reportable in Arizona for many years while surveillance for chronic hepatitis B began in 1998. Hepatitis B vaccines have been available since 1981 but were not widely administered until 1991. The current prevention strategy includes routine vaccination of infants, vaccination of high risk adolescents and adults for infection, and prenatal testing of pregnant women. Because infection at a young age is a significant risk factor for chronic disease, much of the focus in the U.S. has been on infant vaccination. Another strategy is educating infected persons about transmission so as to help contain further spread.

Case definitions for classifying hepatitis B as acute or chronic rely on positive laboratory findings as well as clinical presentation. However, many cases of hepatitis B in Arizona are currently not investigated so classification is based largely on the lab result. A positive IgM result is classified as an acute case; cases are classified as chronic based on a positive surface antigen or envelope antigen test, or nucleic acid testing, in the absence of a positive IgM result.

Surveillance data for hepatitis B reflect the date of report to the health department and not date of infection or onset of symptoms. Acute hepatitis B in Arizona has been increasing since 2001, with the highest numbers reported in 2005 (Figure 12). Reports of chronic hepatitis B in Arizona have been consistently higher than for acute hepatitis B, but do not appear to be increasing.

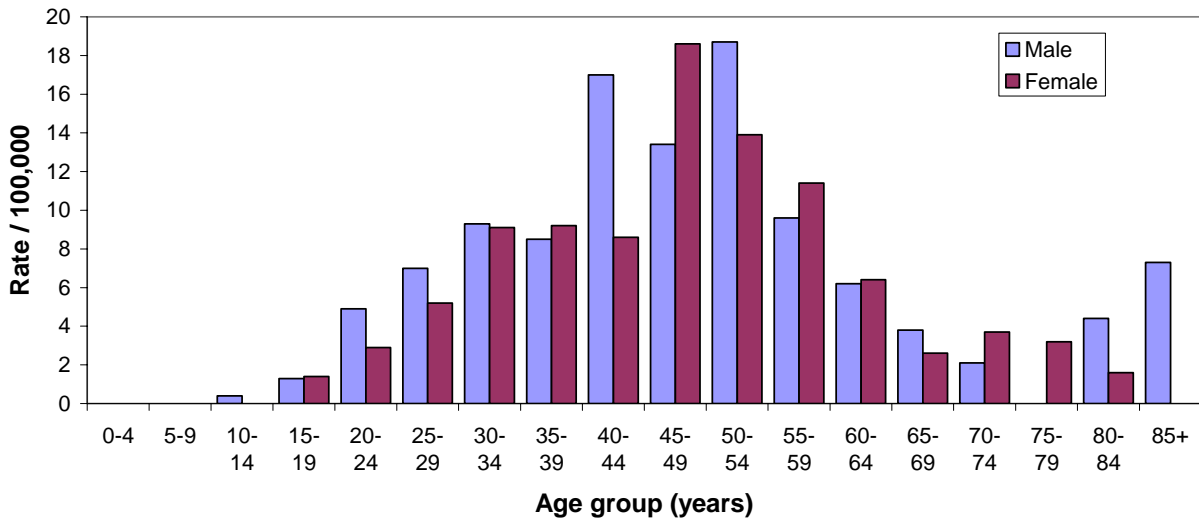
**Figure 12. Rates of hepatitis B by year of report, Arizona and U.S., 1995-2005**



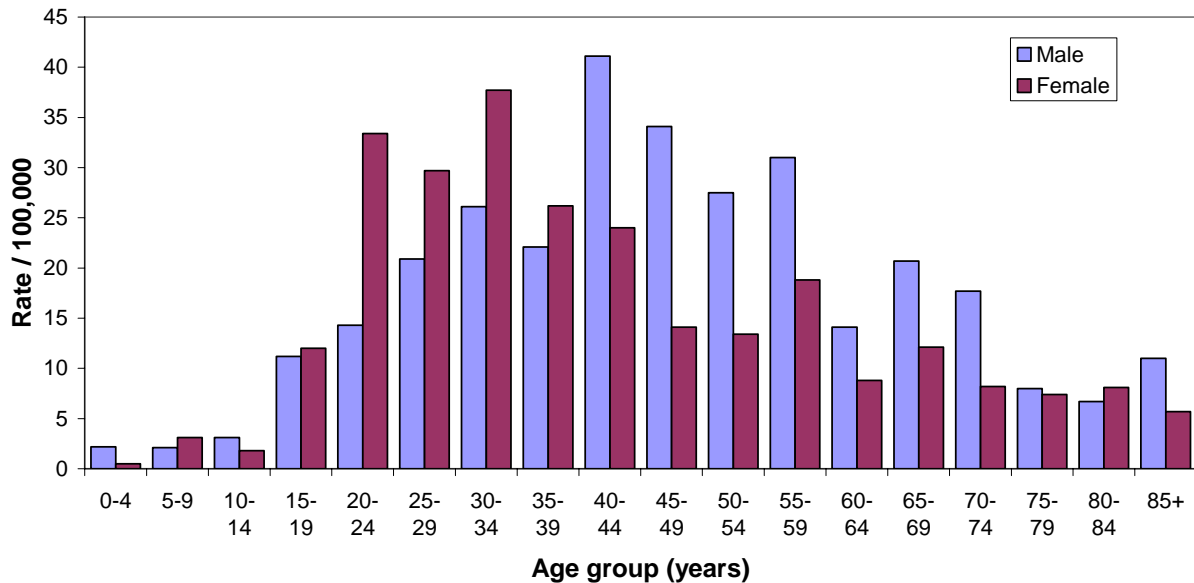
Rates of acute and chronic hepatitis B by age and sex are shown in Figure 13 and Figure 14. (Note the scales are different for these graphs.) Males account for 53% and 52% of acute and chronic cases, respectively. Approximately 55% of acute cases among both males and females are between 40 to 59 years. For chronic hepatitis, approximately 45% of cases among males are in the same age group. However, among females, cases of chronic hepatitis are being identified in a much younger group, with only 27% of cases aged 40-59 years. It has not been determined whether this represents a true discrepancy in the age distribution of cases, or

whether this is a result of increased testing among younger women as a result of prenatal screening.

**Figure 13. Rates of acute hepatitis B, by age and sex, Arizona, 2005**

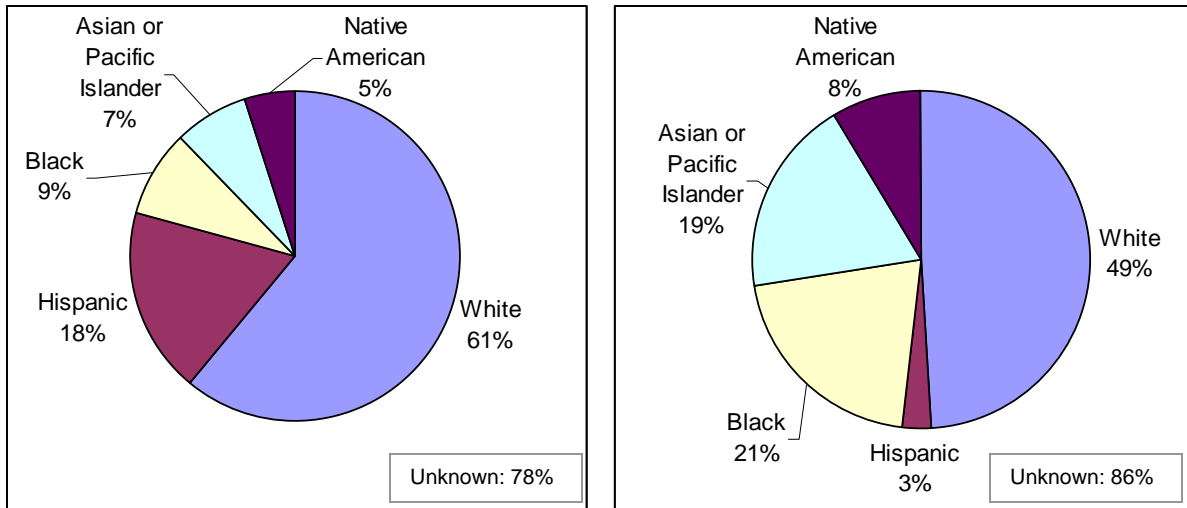


**Figure 14. Rates of chronic hepatitis B, by age and sex, Arizona, 2005**



The racial/ethnic backgrounds of reported cases of acute and chronic hepatitis B cases are shown in Figure 15. While White non-Hispanics and Hispanics are strongly represented among acute cases (61% and 18%, respectively), these two groups make up much smaller proportions of the chronic cases, with Blacks and Asian/Pacific Islanders representing 21% and 19%, respectively. It is important to note, however, that race/ethnicity is unknown for 78% of acute cases and 86% of chronic cases.

**Figure 15. Racial/ethnic representation of reported cases of hepatitis B, acute (left) and chronic (right), Arizona, 2005**



While acute hepatitis B appears to have been increasing in Arizona in recent years, more information is needed about whether this increase represents a true rise in symptomatic cases, or rather an increase in requests for testing, changes in the sensitivity of the tests, testing availability, public knowledge, or other factors. Further investigation of hepatitis B cases regarding clinical presentation, risk factors, transmission, and reasons for testing is needed in order to better understand the epidemiology of hepatitis B in Arizona.

## E. Influenza and Respiratory Syncytial Virus (RSV)

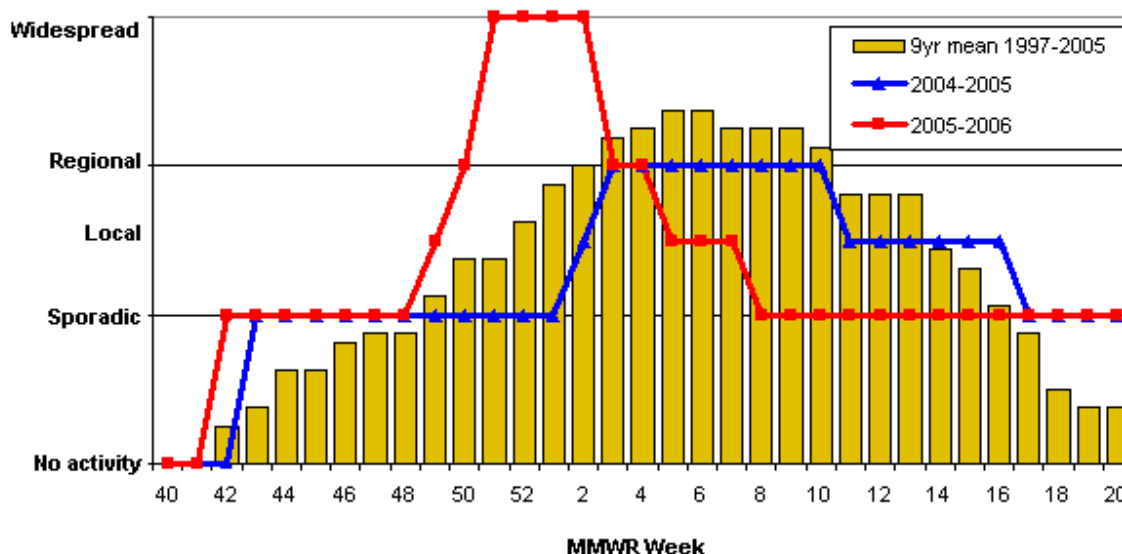
### Influenza

Influenza became laboratory-reportable in October, 2004. While not a reportable disease before this change, influenza surveillance has been conducted for many years in Arizona because of the potential public health impact of the virus. There are several purposes for influenza surveillance: to determine where and when influenza cases are occurring; to determine the predominant types and subtypes circulating in the state; to assess the intensity and impact of activity; and to detect the emergence of novel influenza viruses or unusual events.

Influenza surveillance in Arizona relies on sentinel providers, laboratory reports, subtyping of isolates, and, in some counties, hospital emergency department visits or school absenteeism. Sentinel physicians throughout the state submit weekly reports of influenza-like illness (ILI) to the U.S. Influenza Sentinel Provider Surveillance Network, a collaboration between health care providers, state and local health departments, and the CDC. These reports help to determine the period when influenza-like illnesses account for a larger proportion of patient visits, both statewide and nationally. Viral isolation and subtyping at the Arizona State Laboratory and other select laboratories detect the predominant circulating types and subtypes and identify any novel strains. Laboratory reports provide further indication of relative influenza activity levels. Since the 2003-2004 season, influenza-associated pediatric mortalities have been nationally notifiable.

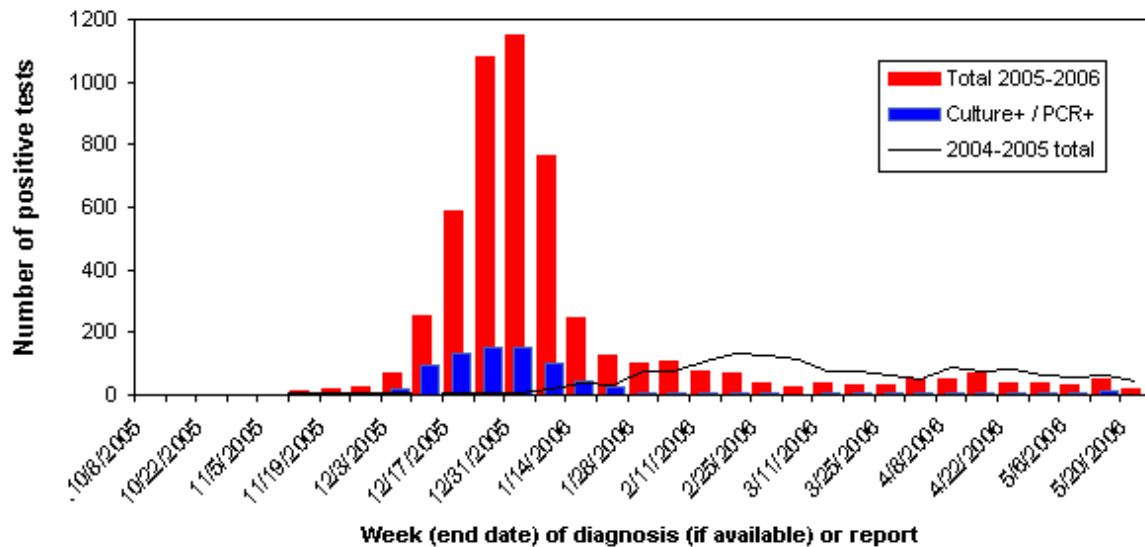
The 2005-2006 influenza season was earlier than usual for Arizona, and with a relatively short but intense peak. Arizona activity was highest in late December and early January; Arizona often experiences peak activity in late January and early February (Figure 16). Influenza activity in the U.S. frequently affects the eastern states earlier than western states. Interestingly, activity appeared to travel west to east this season, with eastern states experiencing activity later than usual.

Figure 16. Influenza activity, Arizona, 1997-2006



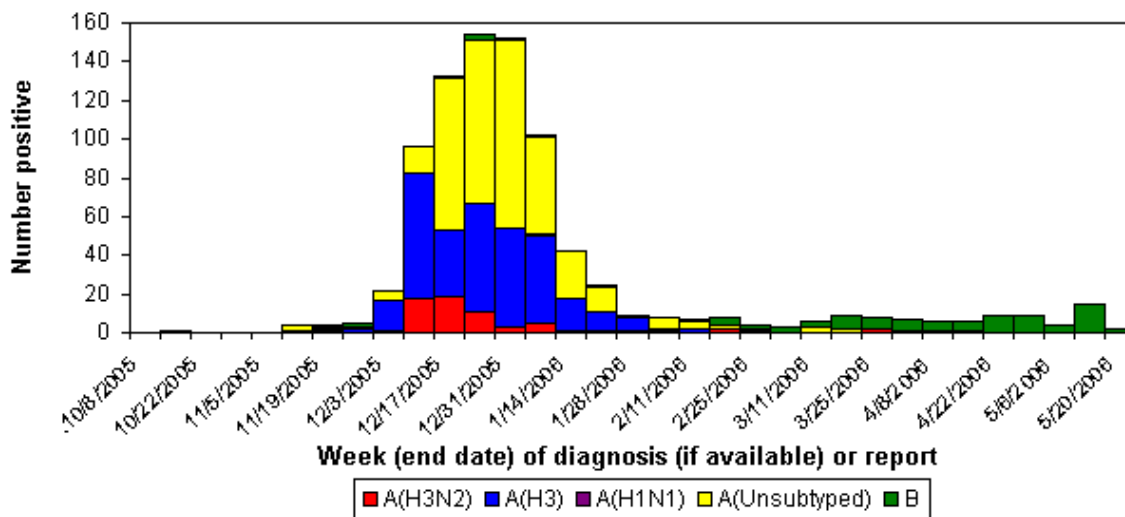
Lab-confirmed reports of flu for the 2005-2006 season are shown in Figure 17. Reporting for the 2005-2006 season was approximately ten-fold the level of the previous year. An assessment of the effects of laboratory reporting on flu surveillance was conducted following the second season of reporting. Lab-reporting was determined to increase the geographic representation of influenza surveillance and has good concordance with data collected through other sources (ILI reporting, state lab data). Labs are already in the habit of reporting other tests routinely to ADHS and in recent years have been doing more and more flu testing; these factors make laboratories a reliable and effective source of influenza information. Lab-reporting has proved valuable for monitoring the timing of activity in the state and identifying counties where the virus circulated.

**Figure 17. Laboratory-confirmed influenza, Arizona, 2004-2006**



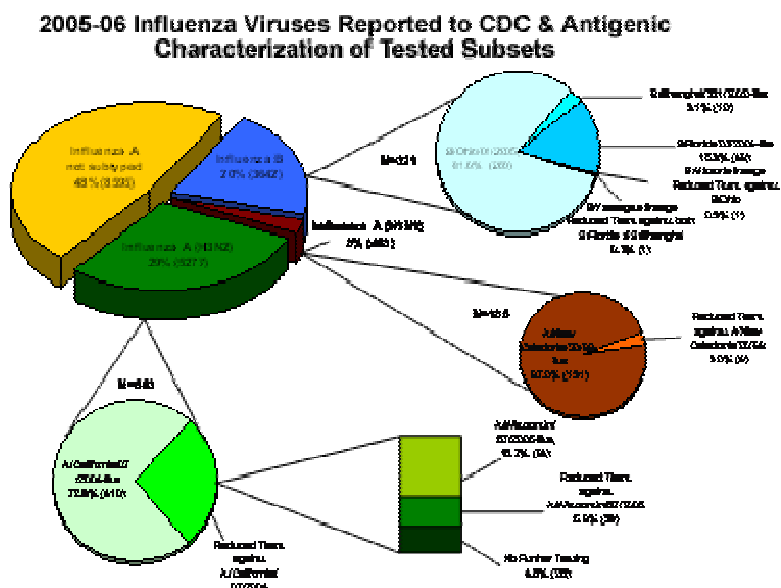
Nationally, influenza A(H3N2) predominated this season while influenza B circulated somewhat later. In Arizona, the peak activity was almost entirely due to influenza A; a small number of influenza B cases were identified late in the season and continued through late May (Figure 18).

**Figure 18. Culture- or PCR-confirmed influenza, by type or subtype, Arizona, 2005-2006**



The three viral components contained in the 2005-2006 influenza vaccine were: A/California(H3N2); A/New Caledonia(H1N1); and B/Shanghai. Nationally, A/California predominated during the season; some isolates which were showing weak reactions to A/California antisera were identified as the newly emergent A/Wisconsin. The antigenic profile for the full season is shown in Figure 19. The World Health Organization and CDC have recommended that the 2006-07 trivalent influenza vaccine for the Northern Hemisphere contain A/New Caledonia/20/99-like (H1N1), A/Wisconsin/67/2005-like (H3N2), and B/Malaysia/2506/2004-like viruses. A/Wisconsin/67/2005 is an antigenic variant of A/California/07/2004.

**Figure 19. 2005-2006 Influenza season antigenic characterization<sup>11</sup>**



Two influenza-associated pediatric deaths were reported in Arizona in the 2005-2006 season. Both occurred late in the season and involved infection with influenza B virus.

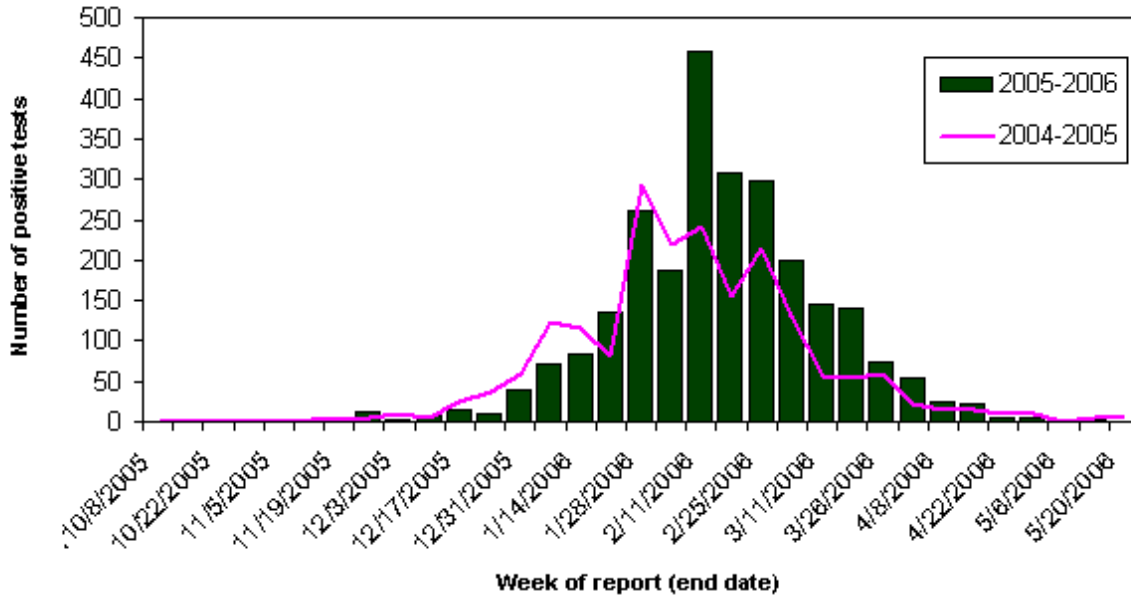
### Respiratory syncytial virus

Like influenza, respiratory syncytial virus (RSV) became laboratory-reportable in October, 2004. Respiratory syncytial virus (RSV) is a common respiratory infection, especially among infants and young children. It follows a seasonal pattern similar to influenza, though peaks during the season often occur at different times. Currently, RSV is tracked as aggregate weekly reports; this may change for the 2006-2007 season, depending on resources. RSV reports during the 2005-2006 season peaked in early February, following the influenza peak, about two weeks later than the RSV peak in the previous season (Figure 20).

<sup>11</sup> Centers for Disease Control and Prevention. 2005-06 U.S. Influenza Season Summary. <http://www.cdc.gov/flu/weekly/weeklyarchives2005-2006/05-06summary.htm>; July 31, 2006.



**Figure 20. Laboratory-confirmed respiratory syncytial virus (RSV), Arizona, 2004-2006**



For the 2005-2006 season, ADHS issued a notice to providers at the beginning of the RSV season—11/29/2005—to inform them that RSV was circulating. During RSV season, RSV antibody injections are administered to high-risk infants to protect against infection. Another notice was issued 4/12/2006 to inform providers that reports of RSV were approaching baseline levels.

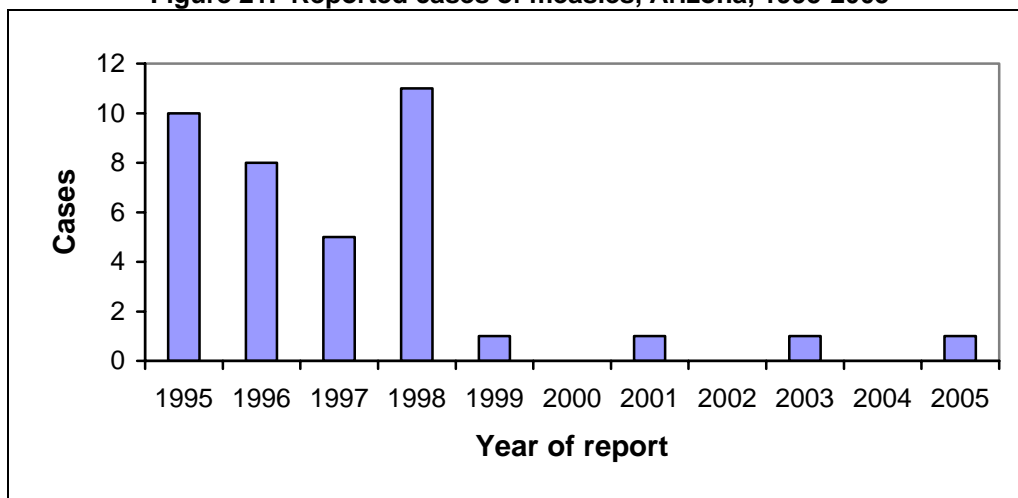
## F. Measles

Measles is a highly communicable viral disease that is becoming increasingly rare in the United States. Measles transmission is through respiratory droplets and airborne aerosolized droplets. Measles vaccine is a live attenuated vaccine and is available as a single antigen preparation, combined with mumps and rubella vaccine (MMR), or combined with mumps, rubella, and varicella vaccine as MMRV. Two doses of measles vaccine are routinely recommended for all children, the first dose given on or after the first birthday and the second dose given at least 28 days after the first dose. In addition, health care workers and students of colleges and universities are required to have evidence of two-dose vaccination and/or measles immunity. According to the 2005 National Immunization Survey results, 93% of Arizona children born from February, 2001 through May, 2003 received one dose of MMR by two years of age.

Interruption of indigenous transmission has been achieved in the United States and other parts of the western hemisphere. Thus, reported cases are likely to be the result of importations. In the United States in recent years, reported outbreaks have been small and have mainly involved high school and college students who are unvaccinated or have received only one dose of measles vaccine.

In Arizona, numbers of reported measles cases have decreased dramatically since 1998, with infrequent reports of single sporadic cases with no secondary spread, most of imported origin (Figure 21). In mid-January, ADHS and Maricopa County Department of Public Health (MCDPH) received a report of suspected measles in a visiting professor who had recently arrived in the United States. The case was later confirmed by serology. The case, who was infected outside the United States, displayed classic measles symptoms: a prodrome of a high fever, cough, and conjunctivitis, followed by a maculopapular rash starting on the face and spreading to the trunk and extremities.

**Figure 21. Reported cases of measles, Arizona, 1995-2005**



MCDPH initiated an investigation and identified close contacts of the case, as well as other contacts potentially exposed at health care facilities and other places visited by the case during the infectious period. The contacts were notified, evaluated for measles immunity, and monitored for measles-like symptoms. The case had visited several locations on the Arizona State University (ASU) campus while infectious. Shortly after confirmation, ASU posted measles exposure notices around campus. A school exclusion policy was implemented for all

students who could not provide documentation of receipt of two MMR vaccinations and were born after 1956. Excluded students were allowed to return to campus immediately after vaccination.

In the three weeks following the initial report of the case, MCDPH ruled out numerous reports of possible measles cases by evaluating clinical symptoms and collecting specimens for lab testing. The exclusion policies at ASU were lifted after three weeks. No secondary cases were identified.

## G. Invasive Meningococcal Disease

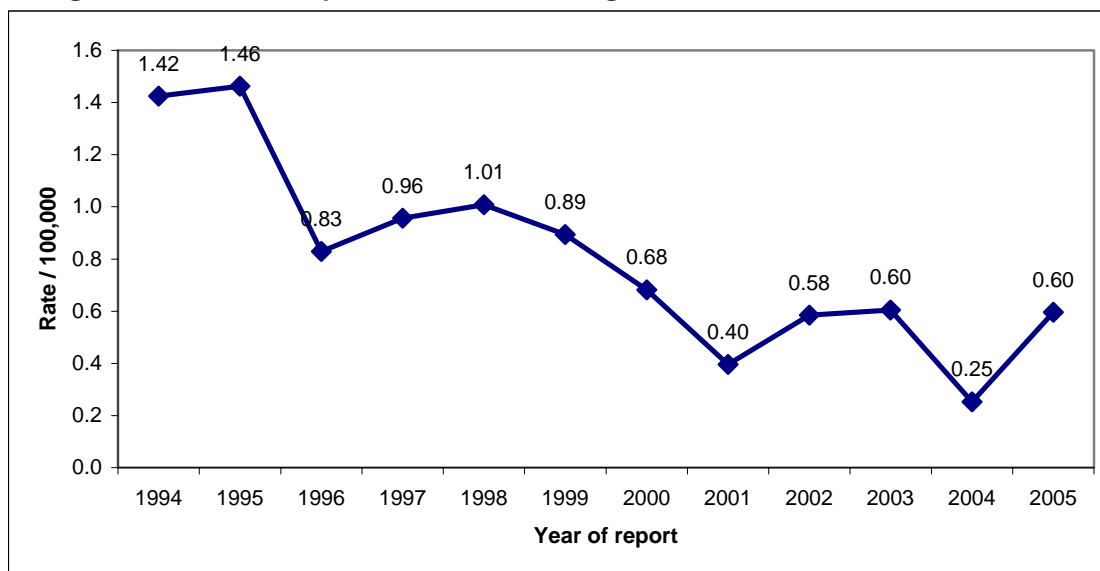
Meningococcal disease is caused by the bacteria *Neisseria meningitidis* and is currently the most common cause of bacterial meningitis for toddlers, adolescents and young adults in the U.S. *N. meningitidis* is divided into numerous serogroups based on immunogenicity, but 95% of illness worldwide is caused by five serogroups: A, B, C, Y and W-135. *N. meningitidis* is spread via respiratory and nasal secretions. Case fatality has decreased with antibiotic treatment; however, it remains high at 10%.

There are now two quadrivalent meningococcal polysaccharide vaccines available in the United States. Both vaccines cover serogroups A, C, Y and W-135. Neither provides protection against serogroup B, which is common in the United States.

The first – meningococcal polysaccharide vaccine – was licensed in the United States in 1978 and is approved for persons 2 years of age or older. The second vaccine is a meningococcal conjugate vaccine that includes a diphtheria toxoid protein. It was first licensed in the United States in 2005 and is the preferred vaccine for persons 11 to 55 years of age. Since licensing of this second vaccine, new vaccine recommendations have been issued for 11- and 12-year-old children, for unvaccinated adolescents at high school entry, and for college freshmen living in dormitories based on increased incidence of disease in these groups.<sup>12</sup>

The reported rate of invasive meningococcal disease in Arizona has largely been decreasing over the past decade, though 2005 increased over 2004 numbers (Figure 22). A total of 36 cases were reported statewide, including one meningococcal death. The fatal case occurred in a 17-year-old male in Pima County. He had not received the meningococcal vaccine and was infected by *N. meningitidis* serogroup Y.

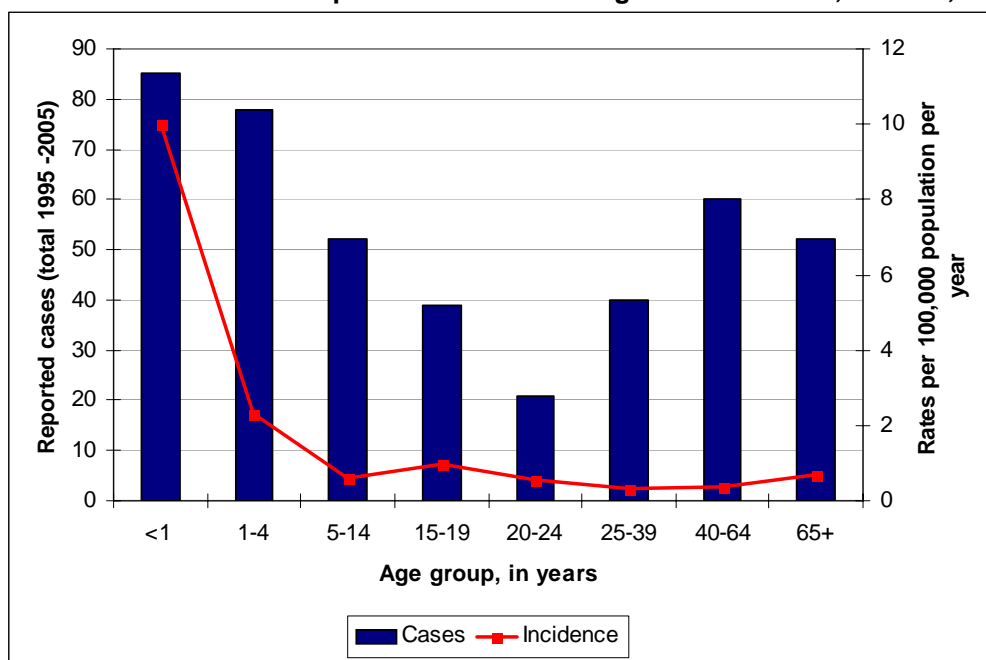
**Figure 22. Rates of reported invasive meningococcal disease, Arizona, 1994-2005**



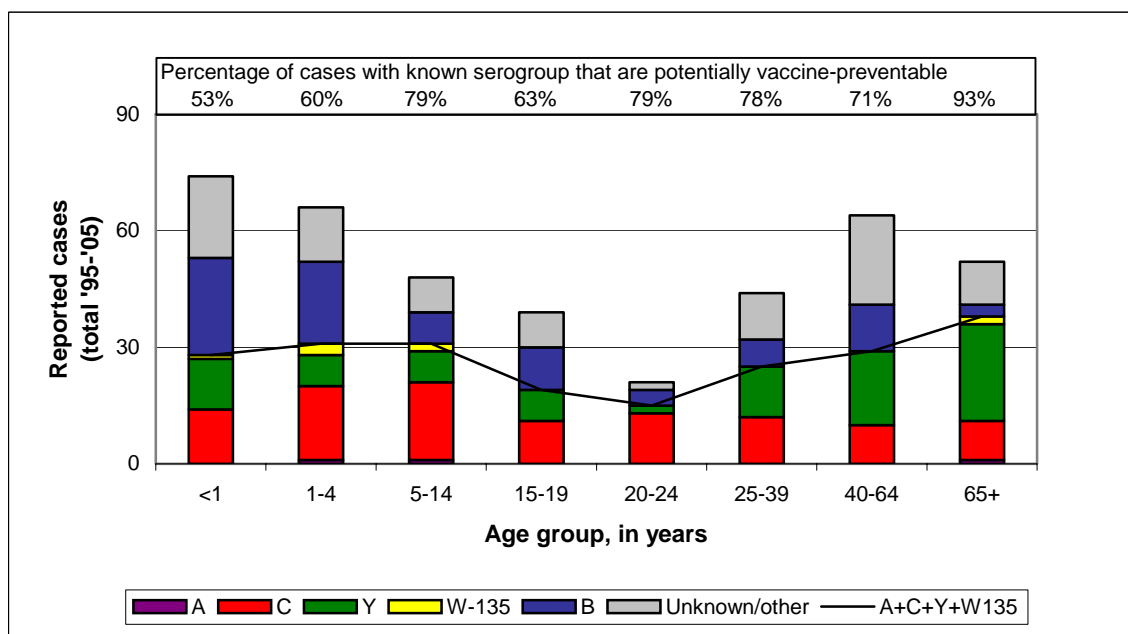
<sup>12</sup> Centers for Disease Control and Prevention. Prevention and Control of Meningococcal Disease Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2005;54(No. RR-7):1-28.

Rates of meningococcal disease vary by age group (Figure 23). The highest incidence rate occurs in children under one year, followed by children ages 1-4 years. A large percentage of disease in those less than one year (47% of those with known serotype in 1995-2005) is caused by serogroup B and thus is not currently vaccine-preventable. Analysis of meningococcal cases indicates that in the past decade over 50% of infections in Arizona were caused by serogroups represented in the current vaccine. As indicated in Figure 24, a high percentage of Arizona cases, especially among adolescents and adults, are potentially vaccine-preventable.

**Figure 23. Rates and cases of reported invasive meningococcal disease, Arizona, 1995-2005**

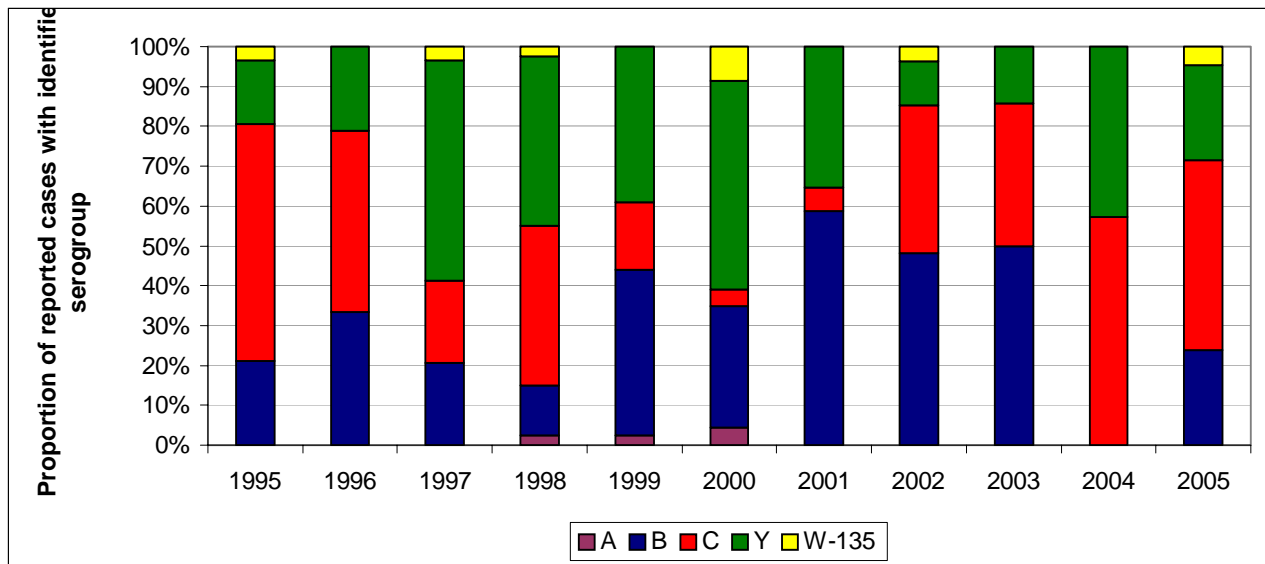


**Figure 24. Serogroup distribution by age group, invasive meningococcal disease, Arizona, 1995-2005**



The serogroup distribution for the years 1995-2005 in Arizona is shown in Figure 25. Nationally, the proportion of meningococcal cases caused by serogroup Y has increased from 2% in 1989-1991 to 37% in 1997-2002.<sup>13</sup> However, no clear trend in serogroup distribution has been observed in Arizona over the similar time frame shown below. In 2005, serogroups B and Y each accounted for approximately 15% of cases with known serogroup; serogroup C accounted for approximately 30%. However, 36% of the reported cases were not serogrouped.

**Figure 25. Meningococcal serogroups, invasive disease, Arizona, 1995-2005**

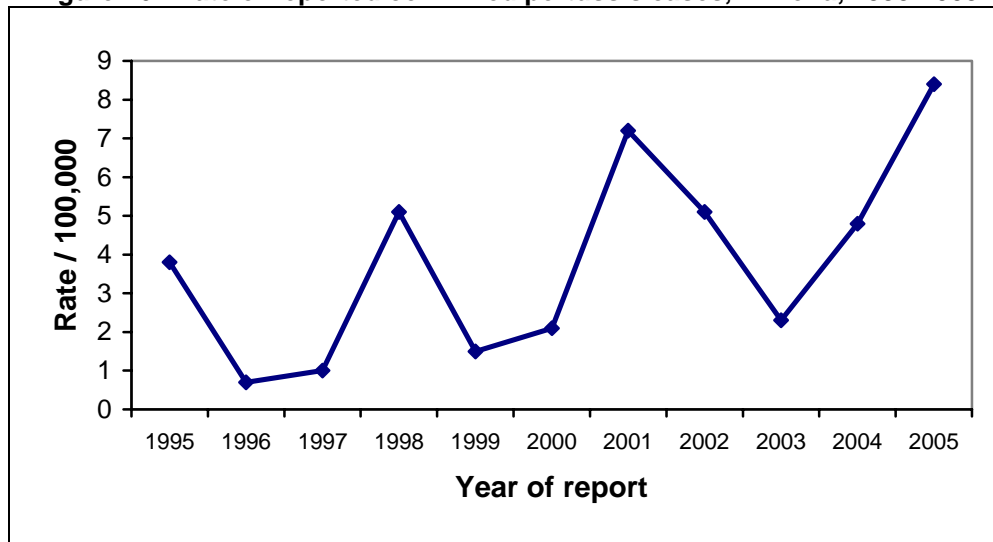


<sup>13</sup> Centers for Disease Control and Prevention. Prevention and Control of Meningococcal Disease Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2005;54(No. RR-7):2.

## H. Pertussis

Pertussis, or whooping cough, is a highly contagious respiratory infection caused by the bacteria *Bordetella pertussis*. Symptoms of the illness begin with runny nose and mild cough, and progress to paroxysms or spasms of coughing, post-tussive vomiting, and inspiratory whoop. Symptoms may last for many months, and fever is minimal throughout the course of illness. Pertussis was one of the most common childhood diseases of the 20<sup>th</sup> century and a major cause of childhood mortality in the early part of the century. Following the introduction of pertussis vaccine in the 1940's, pertussis incidence has decreased more than 97% nationally as compared with the prevaccine era. However, in recent years pertussis incidence has increased again. Arizona experiences the characteristic cyclic patterns of pertussis incidence (Figure 26), but the trend over the last ten years demonstrates a statewide increase as well. Pertussis infection in infants less than 12 months of age may result in more serious illness, more complications and hospitalizations, and higher mortality rates than pertussis infection in older age groups. Pertussis infection among older adolescents and adults is commonly milder or without symptoms, and inspiratory whoop is uncommon in these age groups.

**Figure 26. Rate of reported confirmed pertussis cases, Arizona, 1995-2005**

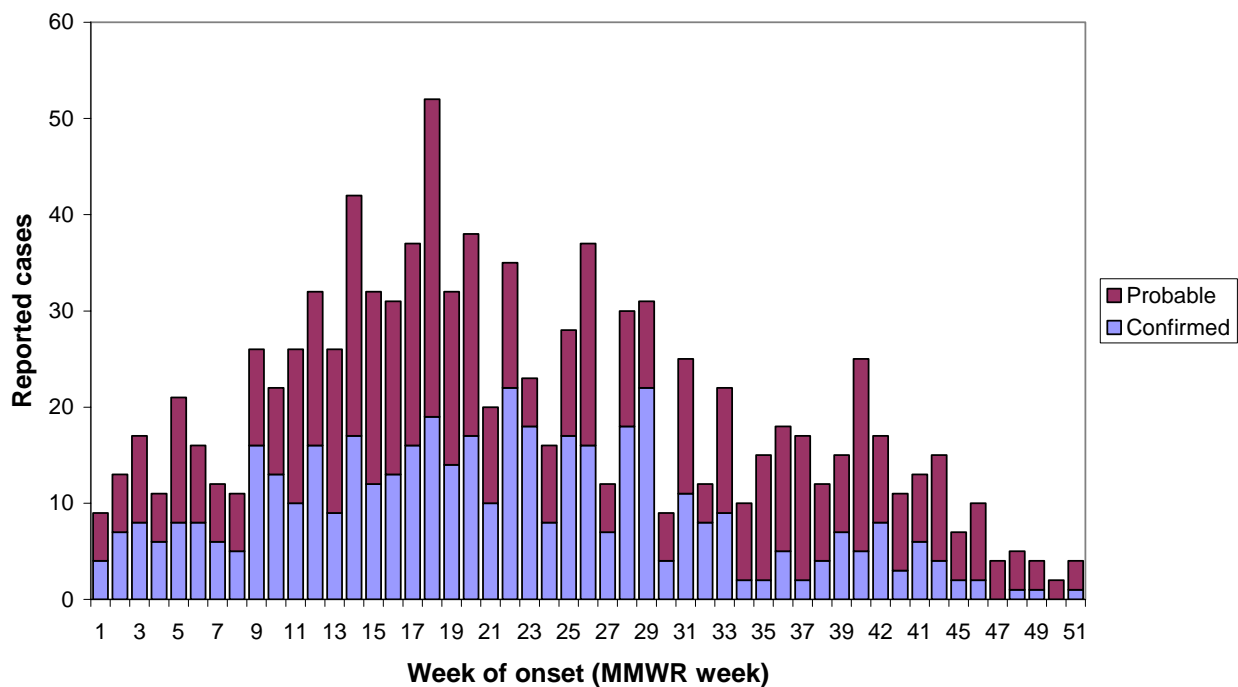


Two pertussis vaccines are currently available: DTaP (pediatric formulation licensed for the primary vaccination series in 1996), and the newly licensed Tdap (adolescent-adult formulation with reduced amounts of pertussis antigens, licensed for use in 2005). Tdap is the first pertussis-containing vaccine licensed for children over 7 years of age in the U.S. In October, 2005, the Advisory Committee on Immunization Practices (ACIP) formally recommended Tdap for adults 19-64 years of age to replace the next scheduled booster dose of tetanus and diphtheria toxoids vaccine (Td), or sooner for adults who have close contact with infants <12 months of age.<sup>14</sup> This vaccine is expected to affect the epidemiology of pertussis nationwide and in Arizona.

<sup>14</sup> Centers for Disease Control and Prevention. Preventing tetanus, diphtheria, and pertussis among adolescents: use of tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccines: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2006;55(No. RR-3):1-46.

In 2005, Arizona experienced significant increases in pertussis activity above baseline levels, and several outbreaks were reported in middle and high schools. On May 19<sup>th</sup>, 2005, a statewide outbreak of pertussis was declared. As a result of the outbreak declaration, a recommendation was made to all providers that administer vaccine to accelerate the childhood immunization schedule so that infants could be protected against pertussis as early as possible. In addition, a more sensitive outbreak case definition was adopted to enhance case finding and intervention, and the newly licensed adolescent-adult pertussis vaccine (Tdap) and antibiotics were purchased and distributed to local health departments to use as needed. These and other outbreak interventions (media campaigns, provider notifications, and rule changes to promote the receipt of Tdap in place of Td for the required school dose), were accompanied by a declining number of reported cases during the summer and early fall months. The statewide outbreak declaration was rescinded in October. Two counties continued to have pertussis outbreaks through the end of 2005 (Pima and Coconino Counties), but in Arizona as a whole, the majority of reported cases had onsets in the spring and summer (Figure 27).

**Figure 27. Onset date of reported pertussis cases, Arizona, 2005**



By the end of 2005, 1,108 confirmed and probable pertussis cases were reported. Over half of the cases were reported by Maricopa County (630 cases, 56.1%) followed by Pima County (320 cases, 27.2%). Rates of reported confirmed and probable pertussis were highest in Coconino and Pima Counties (48.3/100,000 population and 35.1/100,000, respectively) (Figure 28). All Arizona counties had cases of reported pertussis by the end of the year. The majority of reported confirmed and probable cases occurred in adults ages 20 years and older (418 cases, 37% of cases among all ages), followed by children age 10-14 years (207 cases, 19%) (Figure 29). The age data reflect the growing recognition of the importance of pertussis disease among adults and adolescents. Infants less than one year of age (the age group most at risk for severe complications) accounted for 138 (13%) of reported cases.



Figure 28. Reported confirmed and probable pertussis cases, Arizona, 2005

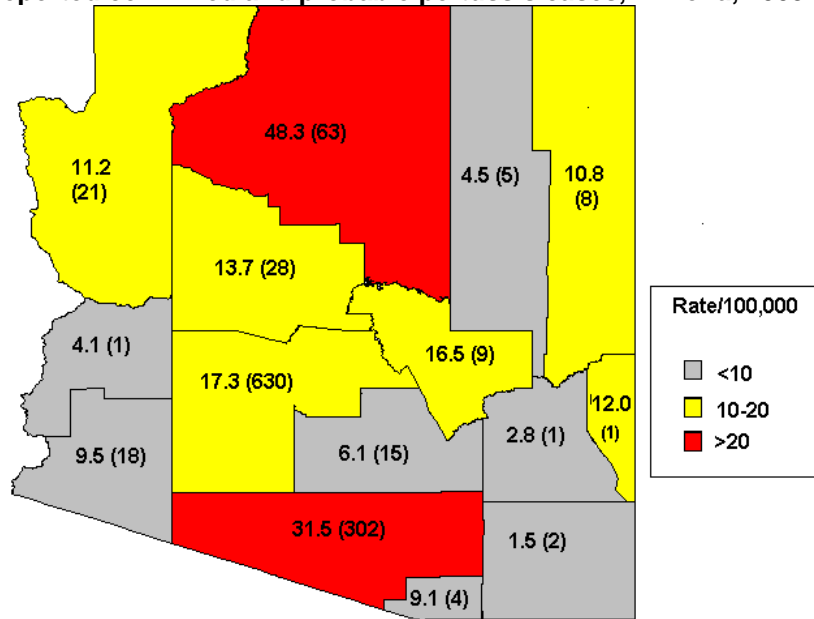
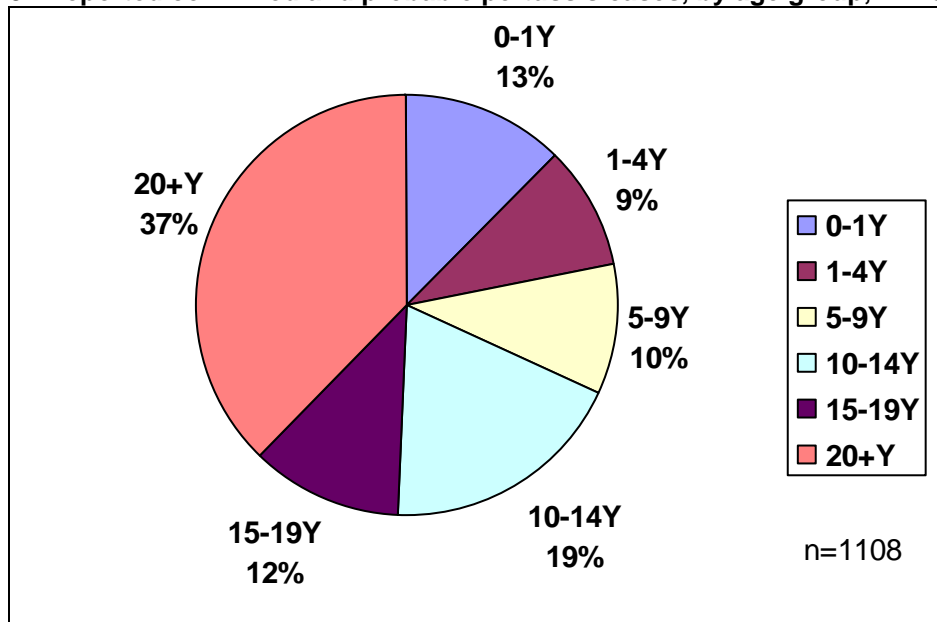


Figure 29. Reported confirmed and probable pertussis cases, by age group, Arizona, 2005



## I. Polio

Indigenous wild type poliovirus transmission has been eliminated from the United States for roughly 25 years. The last reported case of paralytic poliomyelitis caused by endemic transmission of wild poliovirus in the United States occurred in 1979 and the last imported case caused by wild type virus was reported in 1993. Cases of vaccine-associated paralytic poliomyelitis (VAPP) continued to be reported in the United States (144 cases reported in the United States between 1980 and 1999) until a 2000 vaccine policy change recommended the exclusive use of inactivated polio vaccine (IPV) instead of oral polio vaccine (OPV) for all polio vaccination doses in the childhood immunization schedule. The last VAPP case occurred in 1999. However, until polio is eliminated globally, travel may continue to provide a means of bringing poliovirus back to the U.S.

In 2005, the first imported case of VAPP ever reported in the United States occurred in a 22-year old unvaccinated woman who had traveled to Costa Rica (an OPV-using country) as part of a university-sponsored study abroad program. She had been living with a local family for about one month when she visited Colombia for three days. On March 3, two days after returning to Costa Rica, she reported having a sore throat, neck and back pain. Over the next 24 hours, her symptoms worsened to include fever and headache. She was treated at a local hospital for a kidney infection. On March 6, she experienced acute leg weakness and was hospitalized locally and soon transferred to a hospital in San Jose, Costa Rica. On March 9, she was emergently transported by air from San Jose, Costa Rica, to Phoenix, Arizona, for further evaluation.

Upon admission to a hospital in Arizona, the case had bilateral areflexic lower extremity weakness, respiratory failure requiring intubation, and other signs indicative of paralytic polio. Stool specimens collected on March 20 were positive for Sabin strain polio virus types 2 and 3 at the CDC polio reference laboratory; no other enteroviruses were identified. The results of serologic tests for all 3 serotypes were greater than 1:10 for both acute and convalescent specimens. Sixty days after the onset of symptoms, she had residual weakness of both legs. According to the new epidemiologic and laboratory classification of paralytic poliomyelitis cases, this case is classified as imported vaccine-related poliovirus due to onset of illness occurring within 30 days before entry into the United States.

The patient had no history of vaccination with either oral polio vaccine (OPV) or inactivated polio vaccine (IPV). Her Costa Rican host family had young children, ages ~6 months, 3 years, 7 years, and 8 years, although the exposure history provides no clear epidemiological link to an OPV recipient. The case had no underlying medical or immunocompromising conditions.

Polio vaccination is already recommended for persons traveling to polio-endemic countries. However, this case may lead to a change in vaccine recommendations for travelers to countries routinely using OPV. For further details on this case, see "Imported Vaccine-Associated Paralytic Poliomyelitis --- United States, 2005", [MMWR, February 3, 2006 / 55\(04\):97-99](#).

## J. Shiga toxin/*E. coli* Testing

Shiga toxin-producing *Escherichia coli* (STEC) has emerged over recent years as an important cause of gastroenteritis in humans worldwide. STEC infections most frequently present as enteric disease, ranging from mild, watery diarrhea to severe hemorrhagic colitis; 2-7% of infections may result in kidney failure due to hemolytic uremic syndrome (HUS). Serotype O157:H7, the most frequently implicated STEC causing HUS, has been isolated in North America and abroad from large foodborne and waterborne outbreaks, as well as from sporadic cases attributed to animal contact and person-to-person transmission. However, 60 STEC serotypes have been implicated in diarrheal disease, and several non-O157:H7 serotypes have been implicated as the cause of foodborne outbreaks and HUS in the United States, Europe, and Australia.<sup>15</sup>

In the past, many researchers believed that the non-O157 STECs were mainly restricted to locations outside the United States and at very low prevalence in this country.<sup>16</sup> However, several studies such as one conducted in Nebraska in 1997 have demonstrated that non-O157 STEC serotypes are at least as prevalent as serogroup O157 in diarrheal samples in the U.S.<sup>17</sup> As a result, many commercial laboratories have begun using one of the many rapid enzyme immunoassays (EIA) licensed by the Food and Drug Administration for the detection of Shiga toxin in human stool specimens.

Since the onset of this testing methodology, the number of non-O157 STEC pathogens detected in the United States and Arizona has increased substantially. As more commercial and public laboratories expand their ability to detect Shiga toxin in the stools of symptomatic individuals, laboratory-based surveillance for STEC has changed. During 2005, one of the two largest commercial laboratories in Arizona started screening stool specimens for STECs using only EIA. During 2005, 55 cases of STEC were reported, whereas only 28 were reported in 2004. Of the 55 cases reported, 20 were non-O157 cases. Although changes in protocol at private laboratories have made it necessary for the Arizona State Public Health Laboratory to modify its protocol to confirm positive samples, this advance in testing methodology has increased surveillance for STEC. The increased ability to detect non-O157 STECs should lead to better determination of risk factors for all STEC infections.

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<sup>15</sup> Fey PD, Wicker RS, Rupp ME, Safranek TJ, and Hinrichs SH. Prevalence of non-O157:H7 Shiga toxin-producing *Escherichia coli* in diarrheal stool samples from Nebraska. *Emerging Infectious Diseases Journal*. 2000. 6(2): 531-533.

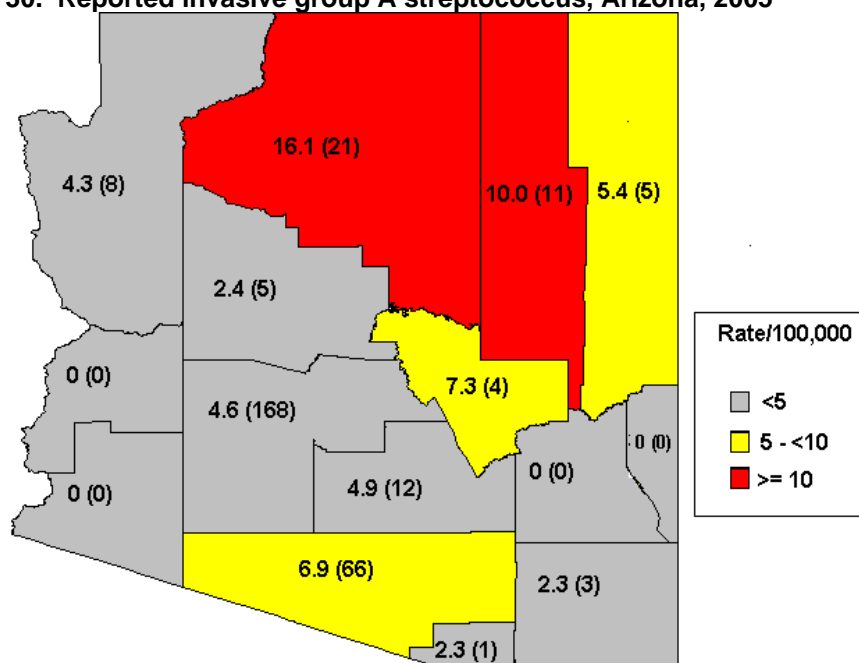
<sup>16</sup> Acheson, D., and G. Keusch. 1996. Which Shiga toxin-producing types of *E. coli* are important? *ASM News*. 62:302-307

<sup>17</sup> Fey, Paul D., R.S. Wickert, M.E. Rupp, T.J. Safranek, and S.H. Hinrichs. 2000. Prevalence of Non-O157:H7 Shiga Toxin-Producing *Escherichia coli* in Diarrheal Stool Samples from Nebraska. *Emerging Infectious Diseases* Vol6.No5.

## K. Group A Streptococcus

Arizona has averaged approximately 220 cases annually of invasive group A streptococcal (GAS) infections for the past decade. In Arizona in 2005, there were 303 cases of invasive GAS infection. The geographic distribution is shown in Figure 30. In addition to the endemic levels of GAS experienced in 2005, an intensive investigation was also conducted in response to healthcare-associated streptococcal toxic shock syndrome (STSS) in Coconino County.

Figure 30. Reported invasive group A streptococcus, Arizona, 2005



Two cases of streptococcal toxic shock syndrome were reported in a Coconino County healthcare facility in February, 2005. Pulsed field gel electrophoresis (PFGE) of the isolates at the Arizona Public Health Laboratory (APHL) indicated that these isolates were genetically related. Subsequently, the isolates were sent to CDC for M protein gene (*emm*) typing. CDC uses a sequence-based system to determine the *emm* gene which encodes the cell surface M virulence protein. Both case isolates belonged to the *emm* type group 1.0, a relatively common *emm* type among invasive streptococcal strains. These isolates were further subtyped at CDC and were identified as *emm*1.6, a rare subtype in the United States.

The initial case was hospitalized with necrotizing fasciitis of the leg and STSS. Her leg was amputated at the hip due to the infection. The second case was a respiratory therapist employed by the healthcare facility and diagnosed with pneumonia and streptococcal toxic shock syndrome. He had cared for the initial case several days after the patient was removed from contact precautions. Both cases survived.

Given the potential transmission in the healthcare facility and upon consultation with CDC and ADHS, the healthcare facility obtained throat swab specimens and questionnaires on all contacts of these cases. Those with Group A Streptococcus (GAS) isolates were given prophylactic antibiotics (azithromycin) to prevent invasive disease. Contacts of any GAS-positive contacts also had throat swabs obtained. All GAS isolates were analyzed by PFGE and compared to the STSS case strain. Isolates were also sent to CDC for *emm* typing.

Throat swabs were obtained from 704 individuals who may have had contact with these two cases. Twenty-one of the “contacts” (2.9%) were positive for GAS. None of the contacts were symptomatic for pharyngitis. Only one of the 21 matched the STSS case strain and *emm* type. This individual did not provide patient care and did not have contact with either case. The period of increased risk for secondary cases of invasive GAS disease is in the first 30 days after exposure. No further cases of invasive GAS occurred during this time period.

Two isolates from previous cases of invasive GAS from Coconino County, one each from 2000 and 2002, were tested to determine if they were related to the 2005 STSS case strain. There was not a match between the 2000 and 2005 isolates. However, the 2002 isolate matched the 2005 STSS strain by PFGE and was further identified to be *emm* type 1.0. The person with the 2002 isolate died of streptococcal toxic shock syndrome.

APHL has now developed the capability to perform *emm* typing of invasive GAS isolates. This laboratory technique will allow for improved investigation of unusual GAS infections.

Persons seeking infection control information should refer to: Prevention of Invasive Group A Streptococcal Disease among Household Contacts of Case Patients and among Postpartum and Postsurgical Patients: Recommendations from the Centers for Disease Control and Prevention. CID. 2002;35 (15 October) 950-95.

## L. Unexplained Death Investigations

Unexplained death with a history of fever became a reportable condition in October 2004, under A.A.C. R9-6-202 & R9-6-377. Reports of this syndrome may come from medical examiners/pathologists, health care providers and hospitals, correctional facilities, funeral home directors and Native American tribes. Cases should be reported to a public health agency within 24 hours of death; investigations will be conducted by the local health departments with assistance from ADHS.

The principle purpose of investigating unexplained deaths with a history of fever is to:

1. Identify deaths of an infectious nature that might be of public health significance;
2. Identify new or emerging infections; and
3. Identify acts of bioterrorism and enhance bioterrorism surveillance.

Reporting and investigation of these cases also enhances infectious disease surveillance of mortalities and facilitates the establishment of communications with nontraditional partners of public health, such as medical examiners' offices.

The case definition for unexplained death with a history of fever follows:

Patient must meet at least one of the following criteria:

1. Hospital/facility-based death with no known cause, with history of fever ( $>38.0^{\circ}\text{C}$ ) within 48 hours of death or a temperature of  $< 36^{\circ}\text{C}$ ;
2. Patient-reported history of fever within 48 hours of death;
3. High clinical suspicion of infectious etiology by health care provider or medical examiner; or
4. Unattended death with no obvious cause of death.

Deaths due to suicide, homicide, trauma or accidents should be excluded.

After it is determined that a case meets these criteria for unexplained death, an investigation is conducted which may include: autopsy; review of medical charts; collection and testing of specimens; or interview of next of kin or other contacts to establish risk information.

Between August and December, 2005, four cases of unexplained deaths were reported in Arizona. Three of the reported cases were in Maricopa County and one in Pima County. Investigations revealed the causes of death for these cases to be:

### ***Toxic shock syndrome caused by the bacterial toxin-producing agent, *Staphylococcus aureus****

In August, a 49-year-old female in Maricopa County died from toxic shock syndrome due to *Staphylococcus aureus*. Symptoms, including nausea, fever, sore throat and progressed to stomach cramps, vomiting and diarrhea, developed on August 25<sup>th</sup> while she was on a cruise ship. Three days later she was seen at an urgent care facility, diagnosed with exudative pharyngitis, and put on antibiotics. On August 30<sup>th</sup> she was taken to a hospital for respiratory distress, where she was intubated, and subsequently developed sepsis with multi-organ failure. She died the next day.

Autopsy results revealed lung and skin involvement. The lungs microscopically showed hyaline membrane disease with patchy areas of exudation with nonspecific inflammation. The skin was lacey on gross examination, with red patches consistent with toxic shock syndrome. Laboratory testing was negative for organisms on blood culture, enterovirus, *Legionella*, *Coccidioides*, rapid strep screen, influenza A and B, and tularemia. Endotracheal respiratory culture grew

*Staphylococcus aureus*. Investigations did not identify any significant exposure and no one on the cruise was ill with similar symptoms. Tissues specimens sent to CDC revealed necrotizing tracheobronchitis from *Staphylococcus aureus*.

### ***Sepsis due to Staphylococcus aureus (one case)***

In October, a 55-year-old female in Maricopa County died from *Staphylococcus aureus* sepsis. She had recently moved to the state and had a history of breast cancer treated with chemotherapy. She was found to be lethargic and vomiting, with blackened mouth and teeth. She called 911 and was taken to the emergency room. While in the ER, she coded and died. Symptoms started three weeks prior with body aches, knee pain and back pain. Two days before visiting the emergency room she complained of nausea, vomiting, fever and a rash on her back. In the ER she had a temperature of 104°F, pulmonary edema in the perihilar region on a chest x-ray, a white blood cell count of 308.9, anemia and low platelets. Blood cultures grew *Staphylococcus aureus* from aerobic and anaerobic bottles. An autopsy revealed disseminated cancer.

### ***Meningoencephalitis***

In September, a 3-year-old male in Maricopa County died from non-polio-enterovirus meningoencephalitis. He reported a two-day history of fever, vomiting and some abdominal problems and was placed on antibiotics. On September 28<sup>th</sup>, the patient went into a local clinic and was transferred to a hospital. He went into respiratory failure in the emergency department, coded and died that day. An autopsy revealed microscopic lymphocytes in the brain tissue and fulminant meningoencephalitis. A nasal swab culture grew non-polio-enterovirus. Tests for West Nile Virus and St. Louis Encephalitis and cultures from other tissues were all negative.

### ***Meningitis***

In December, a 48-year-old male in Pima County was found dead in his residence. He was last seen alive on December 19th. Autopsy revealed acute meningitis with a high number of neutrophils, indicating a probable bacterial etiology for the meningitis.

In 2006, ADHS will continue working with all county health departments and reporting sources to develop protocols and procedures for reporting and investigating an unexplained death with a history of fever.

## IV. Outbreaks





## Background

Foodborne illnesses are a widespread public health problem with an estimated 76 million cases and 5,000 deaths occurring each year in the United States. Health officials in Arizona have several different mechanisms for identifying outbreaks: routine surveillance of reportable diseases and investigations of these cases to identify common exposures and clinical symptoms; routine testing, sub-typing, and comparison of enteric isolates including using advanced molecular identification techniques such as pulsed field gel electrophoresis (PFGE) to detect matching or possibly linked cases; and public reports of suspected foodborne illness to local health departments using foodborne illness hotlines.

### *Definitions:*

#### *Confirmed Foodborne/Waterborne Outbreak:*

A confirmed foodborne illness outbreak is an incident or exposure in which two or more persons experience a similar illness after ingestion of a common food, water source, or meal and epidemiologic evaluation implicates the item as the source of illness. Outbreaks may or may not be laboratory-confirmed. Waterborne outbreaks may be associated with drinking water or recreational water. Confirmed outbreaks may be classified into the following categories:

1. Laboratory-confirmed: Outbreaks in which laboratory evidence of a specific etiologic agent is obtained.
2. Epidemiologically-defined: Outbreaks in which clinical and epidemiological evidence define a likely agent, but laboratory confirmation is not obtained.
3. Outbreak of undetermined etiology: Outbreaks in which laboratory confirmation is not obtained and epidemiologic evidence cannot clearly define an agent.

#### *Probable Foodborne/Waterborne Outbreak:*

A probable foodborne illness outbreak is defined as an incident or exposure in which two or more persons experience a similar illness after ingestion of a common food item or water source, and a specific item is suspected, but person-to-person transmission or other exposures cannot be ruled out.

Healthcare providers report suspected foodborne illness outbreaks when they see an unexpected number of patients with gastrointestinal illness. Restaurants, daycare providers, schools, and healthcare facilities (i.e., hospitals, long-term care facilities) may also report outbreaks.

Norovirus continues to be major cause of gastrointestinal outbreaks in Arizona with 19 confirmed norovirus outbreaks and 14 suspected viral outbreaks reported during 2005. The majority of norovirus outbreaks in Arizona are thought to be spread via person-to-person transmission. Additionally, the handling of ready-to-eat items such as sandwiches, drinks, and salads by an ill food worker can also lead to outbreaks of norovirus. Prevention of further disease transmission occurs by encouraging proper hand-washing techniques, minimizing bare-hand contact with ready-to-eat items, removing environmental contamination, and excluding ill employees from work until 72 hours after recovery.

Bacterial intoxication, or suspect intoxication, caused by such pathogens as *Clostridium perfringens*, *Bacillus cereus*, and *Staphylococcus aureus* was also an important cause of foodborne clusters in 2004. These outbreaks often lack laboratory confirmation since tests are unable to detect the bacteria and toxin which are short-lived in the stool of ill individuals.

Commonly-identified factors leading to bacterial intoxications are improper time and temperature control of potentially hazardous food items such as meat, rice and sauces.

## **Investigations and Outbreaks of Enteric Diseases**

### ***Escherichia coli* O157:H7 Outbreak at Zoo, Maricopa County, July 2005**

In July 2005, two children hospitalized with *Escherichia coli* O157:H7 infections were reported to the Arizona Department of Health Services (ADHS). The positive specimens collected on the children were confirmed at the Arizona State Public Health Laboratory (ASPHL) and tested for relatedness using pulsed field gel electrophoresis (PFGE). The two isolates demonstrated rare, indistinguishable PFGE patterns suggesting a common exposure for both children. Detailed interviews were completed with the parents of both children to determine if a source could be identified.

Although the interviews found the both children had consumed foods of two popular brands, interestingly both had visited the same Arizona petting zoo prior to becoming ill. No common food or beverage was consumed by both children at the zoo and the children's activities outside the zoo were unrelated. During interviews, parents were asked to describe specific activities and exhibits visited while at the zoo. A parent of one of the cases recalled having direct contact with the petting zoo animals in the open interaction area of the petting zoo, while the parent of the second child reported only possible hand contact with the exterior railings at the petting zoo. Although the cases had visited separate exhibits at the zoo on different days, both children had played in an area immediately adjacent to and downhill from the petting zoo facility. Both animals and recreational water sources were tested to identify the source of the infections.

Fecal samples from 15 of 25 (60%) animals and feces from a sheep pen were positive for *E. coli* O157:H7. Individual animals confirmed to be shedding *E. coli* O157:H7 included 12 goats, 2 pigs, and one dwarf zebu cow. Twelve of 15 isolates had PFGE patterns indistinguishable from each other and the two pediatric cases. No environmental samples were positive for *E. coli* O157:H7. Following recommendations from ADHS, the zoo administration closed the petting zoo and adjacent play area to the public and decontaminated the area where the two children played. The petting zoo area was later reestablished in a location with appropriate hand washing facilities and away from other child play areas.

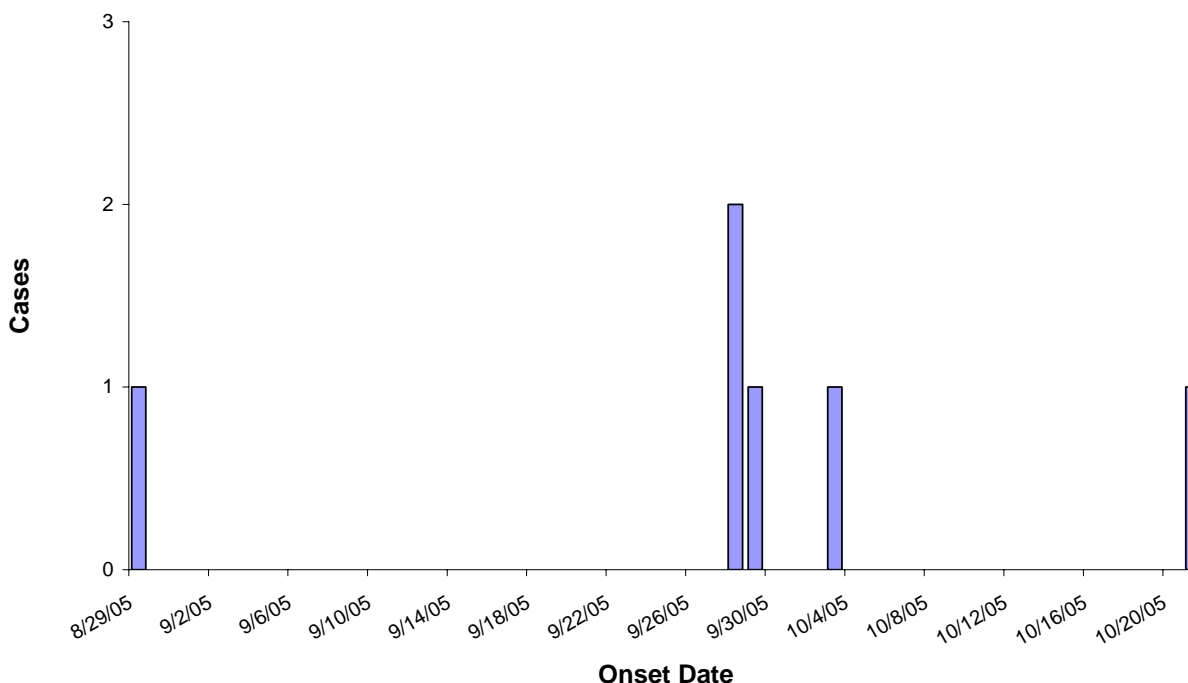
### **Shigellosis Outbreak at Daycare Center, Maricopa County, November, 2005**

On September 20, 2005, the Maricopa County Department of Public Health (MCDPH) received report of a single case of *Shigella sonnei* in a child who had been attending a large, licensed daycare center in a Phoenix suburb during his illness, which began August 28, 2005. MCDPH initiated routine education to the daycare on September 20th prompting the daycare to review current cleaning measures, encourage hand-washing among children and staff, and alert parents and staff of the presence of *Shigella* at the daycare to allow for increased surveillance for other symptomatic children and staff members.

Over the course of the next month, a total of six children aged 3- and 4-years were reported positive for *Shigella sonnei* at the same daycare with onset dates shown in Graph 1. After the report of each case, verbal and written education was provided to the daycare to prevent

additional spread. Three inspections from Maricopa County Environmental Health were completed. In addition, community health nurses (CHNs) with MCDPH visited the daycare on November 3, 2005, distributing written information on germs, hand-washing, cleaning solutions containing bleach, and other educational materials. During the site visit, the CHNs determined that the bacteria were likely spreading due to the children's lack of proper hand-washing. The toddlers appeared be unaware of proper hand-washing techniques or simply did not wash their hands after leaving the bathroom. Also, there was no soap in the dispensers in the 3-year-olds' bathroom. In addition, the children did not stop to wash their hands when returning to the classroom after playing outside during recess.

**Figure 31. Shigellosis cases associated with a Maricopa County daycare, by onset date, August to October, 2005**



After observing the conditions at the daycare center, the CHNs made several additional recommendations: posting information sheets on *Shigella* on classroom doors for two weeks following each confirmed case; arranging for a health educator to visit the daycare and demonstrate proper hand-washing techniques to the staff; sanitizing the toys used in the daycare daily until the outbreak was over; and checking the soap dispensers regularly. According to discussions with staff members after the site visit, it appeared that the education and recommendations presented to the daycare earlier had not been completely adopted.

### **Norovirus Outbreak at Guest Ranch, Pima County, August 2005**

On Wednesday, August 3, 2005, Consumer Health and Food Safety at the Pima County Health Department was notified of a possible foodborne outbreak at a local guest ranch. Inspectors initiated an investigation at the facility the same day, identifying 20 guests and staff ill with diarrhea and vomiting the preceding day, approximately 30-60 minutes after the lunch meal. Symptoms resolved 24 hours after onset. According to the property manager, only two

employees had reported illness during this time – a hostess and a medical staff member – but their onset dates were similar to the ill guests. Employees at the ranch eat the same food as the guests and all guests dine in the main dining room. An inspection of the kitchen area yielded no violations.

In order to determine the cause of the outbreak, Pima County Health Department Disease Control administered questionnaires to guests and staff inquiring about food consumed and daily activities, and collected stool specimens for viral and bacterial testing. Thirty-one employees and 34 guests reported symptoms consistent with the case definition developed by the Pima County Health Department for this outbreak:

“Any guests or staff ill on site at the guest ranch from Sunday July 31, 2005 to Tuesday August 2, 2005, whose symptoms included diarrhea and/or vomiting with a rapid onset and lasting at least 12 hours, with duration of illness not exceeding 48 hours in length.”

Once data from questionnaires were analyzed, no individual menu item or common meal could account for the presence of symptoms. However, the questionnaires did reveal an ill staff member with an onset of July 31, 2005, approximately 48 hours prior to the onset for the rest of the symptomatic staff and guests. This employee became ill prior to the start of his/her shift and continued to work at the guest ranch while symptomatic. This staff member complained of uncontrollable diarrhea, abdominal cramps, and fever. The staff member had contact with staff and guests as well as shared equipment and surfaces, providing a source of infection for those that subsequently became ill.

The Pima County Health Department (PCHD) recommended thorough cleaning, restriction of ill employees, and education on methods to prevent the spread of norovirus. The stool specimens collected from one guest and one staff member tested positive for norovirus GII.4 at the Arizona State Public Health Laboratory (ASPHL). These results are consistent with the symptoms and epidemiology observed. Consumer Health and Food Safety encouraged sanitizing all areas of the facility and ill staff were asked to remain off-site until 72 hours after symptoms resolved. PCHD also provided the property manager with education on norovirus, including modes of transmission and ways to decrease spread.

### **Norovirus Outbreak at Camps Held at a University, Coconino County, July 2005**

On July 20, 2005, the Coconino County Health Department (CCHD) received a call from the Emergency Department at a local hospital reporting three patients ill with vomiting, diarrhea, and dehydration. All three patients were attending an athletic camp on a university campus, two camp attendees and one staff member. Administrators reported that the athletic camp was a two week, onsite training camp with over 200 teenagers from various states. CCHD contacted the athletic camp organizers and discovered that 40 additional athletic camp participants were ill with similar symptoms. The next day, CCHD interviewed several campers.

A total of 115 cases were identified as part of this outbreak. Most of these were attendees or staff at the athletic camp, but university employees and students were also found to be ill with similar symptoms. CCHD determined risk factors for infection included close proximity to someone who was vomiting (potentially through aerosolization of the virus), and contact with contaminated surfaces shared in the dormitory, athletic facility, or other campus locations.

Stool specimens collected from ill campers tested positive for norovirus at the Arizona State Public Health Laboratory (ASPHL). Sequencing of the virus at ASPHL determined that this specific sequence had not been detected in Arizona before, suggesting that it was brought to the camp by a participant who had been infected elsewhere.

In order to control the outbreak, CCHD recommended that all participants of the affected summer camps be confined to their residence halls while ill and that camp attendees and employees practice thorough and frequent hand-washing with soap and hot water. CCHD advised the university to carefully clean and disinfect the affected residence halls, cafeteria, and other contaminated facilities. The university decided to cancel all remaining summer camps on campus to stop the spread of the virus. No additional cases of norovirus illness connected to this outbreak were reported after July 28<sup>th</sup>.

### **Confirmed Case of Hepatitis A in a Food Handler – Yuma County, June 2005**

On June 4, 2005, a local hospital confirmed that a food worker involved in the preparation of ready-to-eat foods had been diagnosed with an acute hepatitis A infection. While the patient did not report any diarrhea or jaundice, the patient felt acutely ill and was hospitalized. The case had elevated liver enzymes and fatigue with onset of symptoms on June 2, 2005.

The food handler was excluded from work and all co-workers were provided hepatitis A immunizations and immune globulin. Although the case did not report any diarrhea, the restaurant had received a variance which allowed bare hand contact with food creating a perception of increased risk. Therefore, immune globulin was administered to patrons who ate at the restraint between May 23 and June 6, 2005. No secondary cases of hepatitis A were identified from this exposure.